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THE TREATMENT AND PROGNOSIS OF CARCINOMA OF THE LARYNX.*

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TOKYO.

The secretary of the American Laryngological Association has honored me with an invitation to speak before this illustrious assembly. I have decided to speak on "The Treatment and Prognosis of Carcinoma of the Larynx," a subject which I have specially studied for the last twenty years.

As you know, the history of the treatment of this particular disease is a long one and the literature on this subject is so extensive that I must forego entering into it. We know, however, from Sendziak's laborious literary studies that, already twenty-five years ago, four periods could be established with

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regard to the views on the treatment of laryngeal cancers: that of therapeutic nihilism, the birth of a rational therapy, and the period of wavering views having been passed through in succession, while at that time one could speak of a period of sober criticism. Since that time the therapy of laryngeal cancer has been further advanced on the one hand by a better diagnosis of its earliest forms and on the other by valuable and almost perfect modifications of the surgical methods, so that we may be justified in considering our present time that of achievement in the treatment of this malignant disease. Thus Felix Semon (London) was able to say in a lecture, which was held not many years before his death, before the Japanese Medical Congress, that carcinoma of the larynx, if diagnosed in its early stages, was curable in 80 per cent of the cases by laryngofissure and excision of the soft parts, a method which your countryman, Solis Cohen, has applied with very good results. Also Gluck (Berlin) is said to have obtained excellent results, even in rather advanced cases, by his applied modification of Billroth's total extirpation, first experimentally tried and later practically in over 300 cases. In a lecture given at Vienna in 1913 he was able to state that in a series of 86 operations he had only one case ending fatally, while in another series of 75 not a single patient died. The results of these two renowned surgeons seem to leave nothing to add to the surgical therapy of laryngeal carcinoma by giving our experiences and studies. But other writers give less favorable reports. Especially the Iitalian authors Gradenigo, Egidi and others would prefer total extirpation of the larynx instead of the thyrotomy recommended so warmly by Semon and Chiari. As to the total extirpation of the larynx, the results of different surgeons have been various. Many, though following closely Gluck's method, have obtained less favorable results, which was already confirmed by the reports of N. H. Pierce, W. Freudenthal and others here in America. Moreover, it seems to me that general surgeons who take an interest in cases of laryngeal carcinoma but being less acquainted with the smaller special laryngeal operations, as, for instance, with the endolaryngeal, are too much inclined to resort at once to the great total operation, even in early cases. On the other hand, laryngologists often delay the total extirpa-

tion, notwithstanding proper indications for it. For these reasons I do not deem it superfluous to take up this question once more, though Otto Chiari thirteen years ago, at the annual general meeting of the American Laryngological, Rhinological and Otological Society in Atlantic City, spoke on the same subject in detail. During more than twenty years of academic and practical activity I have employed various methods in operating on 141 cases of laryngeal carcinoma, and the results obtained and the facts observed have led me to views differing somewhat from those of the above mentioned authors.

First of all, I will speak on the surgical methods which I used and the results obtained, following up with a comparative criticism of other methods taken from the literature on the subject, and ending up with some general conclusions on the subject of laryngeal carcinoma.

The methods employed by me were five in number, viz.: (1) Endolaryngeal excision, (2) thyrotomy and excision of soft parts, (3) partial extirpation, (4) semilateral extirpation, and (5) total extirpation of the larynx.

(1) THE ENDOLARYNGEAL OPERATION.

This method was first employed by B. Fränkel with good results on laryngeal cancer, but on account of uncertain results was rejected by most surgeons. Felix Semon was of opinion that early forms of laryngeal cancer, which according to Fränkel may be treated by an endolaryngeal operation, could be better and more safely treated with laryngofissure, and Gradenigo, Otto Chiari, Ch. Jackson, etc., also positively rejected this endolaryngeal method for the surgical treatment of cancer. Gradenigo was even of opinion that the cases of successful operation had not been cancerous at all and that the time of postobservation had been too short. Being a pupil of Fränkel, I am still of opinion that the earlier a beginning laryngeal cancer is diagnosed, and the more certain the diagnosis is as regards the purely local form of the affection, the more certain this small operation may be successful. This will not only preserve the physiologic functions of the organ and avoid a deformed outward appearance, but will also add

to the fame of the surgeon as to his surgical dexterity. And if a properly diagnosed very small carcinoma happens to be quite local, especially on the free edge of the vocal ligament, the endolaryngeal operation can be resorted to with good prospects. I performed it 23 years ago for the first time on a patient from Kumamoto, 56 years of age, who on the free edge of the right cord had a ragged polypoid cancroid, the size of the tip of the little finger, which had not yet affected the ventricular membrane. The diagnosis was verified by microscopic examination of a testpiece and I accordingly performed the endolaryngeal operation in two sittings within two weeks, with such good result that the patient could be discharged from the hospital after two months. One and one-half years later I presented this man at the medical congress in Tokyo, together with the microscopic slides made before the operation. At this time the right cord showed granular regeneration and produced a good voice. Since that time I have performed the same operation on 15 patients, in whom careful examination showed only strictly localized polypoid forms of cordal cancer, not yet spreading to the ventriculum, and also on two cases with pedunculated ulcerous forms of larynx carcinoma on the free edge of the epiglottis. The former were mostly performed under the laryngoscope with the aid of the sharp larynx forceps, or double curette; in only three cases "Schwebelaryngoscopie" was applied. The pedunculated forms were operated under the laryngoscope with the galvano-caustic loop, as shown by Loeb in his work on surgery, and the cancers at the end of the epiglottis also with the aid of the laryngoscope and the double curette. The results were on the whole very good. In five cases repeated examinations showed a complete cure, viz. the first patient, who died 18 years later at the age of 75 of influenza pneumonia, was during all these years well and showed no signs of recurrence. The second case with right sided cord epithelioma remained well for 11 years, the third with left sided cord cancroid for eight years, the fourth with pedunculated carcinoma on the left aryepiglottic fold and the fifth with right cord epithelioma five years later was quite healthy and free of returnation. The other 11 endolaryngeal operations showed a relative cure; the patients left the hospital almost well, the scar was healed and no local relapse or gland-

ular metastasis was visible. Half of them, which could be kept under observation, were well after more than a year. They were all exhibited at the monthly meetings of the Japanese Otolaryngological Society. Only in three cases, the results were not good; one case with medullary carcinoma of the left cord, which besides the local symptoms of a sharply outlined superficial and ragged polypoid growth on the middle of the left cord already showed a slight affection in motility of the left cord, had to be treated a second time endolaryngeally a month later on account of recurrence and, two weeks later, total extirpation of the larynx became necessary. The other two cases, one of carcinoma pendula on the tuberculum epiglottica and another of right cord cancroid soon showed local recurrence, but further treatment was refused.

With the above results of my endolaryngeal operations I have been well satisfied, but unfortunately in the majority of cases of laryngeal carcinoma the operation can not be resorted to, because when the patients come to us it is already too late. I must emphasize that the indication for this minor operation must strictly be that the tumor is sharply defined in a restricted place. In every case of such nature I am not satisfied with the ordinary laryngoscopic examination, but I try to examine the ventricular space with my ventricular laryngoscope in order to see if the ventricular arch is already affected or not, and besides any possible affection of motility of the vocal ligament with the aid of the mirror. These two points I consider of the utmost importance to ensure the possibility of a successful endolaryngeal operation. The affection of the membrana ventricularis is a sign that the carcinoma is no longer limited to the edge of the ligament, and a diminished motility of the cord, which according to Felix Semon is a very early symptom of laryngeal cancer, is in my opinion proof that the cancerous infiltration is no longer superficial, but has already penetrated into the cord muscles or to the cricoarytenoid joint. As to the technic of the operation nothing new need be added. I would only recommend that, after the tumor has been removed by any of the aforesaid methods, the spot should be thoroughly cauterized with the galvanocautery. Local radium application after the operation, which has lately often been applied for prophylactic purposes, seems to have been beneficial.

(2) THYROTOMY.

This method is said to have been first used by Solis Cohen in 1868 for laryngeal carcinoma, but has come into vogue only later on the recommendations of Felix Semon, Otto Chiari, Hansberg, Ch. Jackson, etc., in the early forms of laryngeal cancer. Felix Semon, indeed, prefers this thyrotomy with the excision of the soft parts to the endolaryngeal operation recommended by Fränkel under similar indications, as not being dangerous and prognostically much better and much more favorable for the preservation of the voice. He was also of opinion that the estimation of the extent of the infltration could not always be properly ascertained by laryngoscopy, and the excision of the tumor by the endolaryngeal operation is not always sufficient. But I still maintain, that a smaller operation should, if possible, be resorted to, if it can be presumed that all diseased parts can properly be removed. Thus, I have had little opportunity to use thyrotomy, because I have employed the former method, where other authors resorted to thyrotomy. It is only when the cord carcinoma had, notwithstanding its early stage, already penetrated into the ventricular membrane or to the ligamentum thyreoarytenoideum or motility disturbances had already appeared that I performed this operation in 12 cases in all. Four of these were cancroids of the right or left vocal cord, three adenocarcinoma of the ventricle, two cancroid of the vocal cord and ligamentum thyreoarytenoideum, and one case each of medullary cancer of the left cord and adenocarcinoma of the left ligamentum thyreoarytenoideum. The results of our operations were not so good as those reported by Semon and Chiari. Five cases showed local recurrence, two of which were afterwards treated with total extirpation and the remaining three were lost sight of; two cases ended fatally, one from infectious pneumonia, the other on account of general debility after a long treatment for granulation proliferation on the wound. Only two cases proved entirely cured (one for over five years, adenocarcinoma of the left ventricle, another for over two years, cancroid of the right cord). The remaining three cases left our hospital in good state, but could not be kept under observation. The reason that my cases of thyrotomy gave inferior results is prob-

ably that they were much more unfavorable from the beginning, for the lighter cases were all treated endolaryngeally, where others would have applied thyrotomy. Under these circumstances I consider my results fair enough, considering that there already had been extensive infiltration, and yet two definitely and three relatively cured cases could be shown. The technic of the operation does not call for any remarks here, except that thyrotomy was always easily performed with cocain or novocain infiltration, sometimes combined with subcutaneous injection of pantopon scopolamin solution, and that the bleeding in operating the cordal carcinoma was never severe, so that I never was obliged to ligate the arteries or to apply Trendelenburg's or the sponge canula. I always performed tracheotomy shortly before the operation proper and the wound of this was always provided with the ordinary tracheal canula, which was removed after one week. The laryngeal cavity after the splitting of the thyroid cartilage was immediately painted with cocain adrenalin solution, and tamponed with iodoform gauze for a day after the operation. The further treatment was without tamponade.

(3) PARTIAL RESECTION AND (4) SEMILATERAL EXTIRPATION OF LARYNX.

Both methods were formerly frequently resorted to in the treatment of laryngeal cancer, seldom with good results. I have, therefore, always been of opinion that sharply defined semilateral or localized cancers, as such of the epiglottis, which had penetrated rather far, but which appeared as if easily removable by semilaryngeal or partial resection of the larynx, should, from the point of prognosis, at once be totally extirpated, because in both procedures the prophylactic precautions against pneumonia are insufficient, and the inclination to recurrence is very marked. For these reasons I have found few occasions to make practical use of any of these methods. But induced by Gluck's report, which he made in 1913 at the meeting of German naturalists and physicians in Vienna, that in 49 cases of hemilaryngectomy he had only three deaths, but 21 complete cures, I used this method in two well defined cases of semilateral cancer, but unfortunately lost both, one by pneumonia after one week, the other through recurrence after five

months. Partial extirpation I performed three times, twice for carcinoma of the epiglottis by using subhyoid pharyngotomy. Of these one, the last was successful, in which I, after removing the epiglottis tumor, sewed the wound surfaces of the basis of the tongue and the aryepiglottic folds together to prevent pneumonia. After three months, by a small reoperation, communication between the pharynx and the larynx was again established and the outer subhyoidal opening closed. The other two cases, in which after extirpation of the epiglottis tumor I closed the outer wound with sutures, ended fatally through pneumonia. These experiences confirmed me in my opinion of the impracticability of hemilaryngectomy and partial extirpation of the larynx.

(5) TOTAL EXTIRPATION OF THE LARYNX.

Notwithstanding the good results of thyrotomy and laryngofissure obtained by such renowned specialists as Felix Semon, O. Chiari, Ch. Jackson and others, and of the endolaryngeal operation by Fränkel and myself, they can only be used in a limited number of cases, as cancer of the larynx usually comes to the specialist in already relatively advanced stages. Therefore, the principal operation will always remain the total extirpation of the larynx, as only in this way can all pathologic processes in the larynx be radically removed, and by further improvement of this method the inoperable cases may in future be more and more lessened in number. After Billroth first tried it in 1873, many famous surgeons followed him, but as you know, the results of these operations were at first not at all good, though, as Sendziak's statistics show, the fatal cases have become fewer in the course of time, and some complete cures were even established by this method (Obalinsky, Solis Cohen, etc.). This, on the one hand a pessimistic view came into vogue, which, supported by statistics, repeatedly asserted, that humanity would lose nothing by giving up this method, which was not to the credit of surgery; on the other hand, it is undoubtedly true that in recent times the total extirpation of the larynx carcinoma is quite frequently practiced by surgeons and by many laryngologists and that the results have become increasingly better. This progress has no doubt gone hand in hand with that of aseptic surgery in general, but

for the progress in this special line we are chiefly indebted to Gluck of Berlin. In 1881, at the Surgical Congress in Berlin. Gluck published his experimental treatises, in which he showed that, in extirpating the larynx, or to express it more generally, in all operations, which so often cause death by pneumonia through foreign bodies entering the air passages, an absolute guaranty against this form of pneumonia could be established by prophylactic resection of the trachea and circular sewing up of the central stump of the trachea to the skin. In the years following he practiced this method with excellent results on many patients, and thus Gluck's method of total extirpation of the larynx has been brought to its present standard of excellence. The principal procedures of it are: (1) Complete exposure of the larynx before opening the air passage; (2) severing the larynx from the pharynx; (3) immediate closing of the pharynx defect while the larynx is drawn far out of the wound; (4) severing the larynx from the trachea; (5) circular sewing in of the lower stump of the trachea into the skin wound, and (6) closing of the whole wound by sutures.

Nineteen years ago I tried this method for the first time, but an unexpected difficulty made me modify Gluck's method to some extent, which, however, also gave satisfactory results. The patient was a merchant, 51 years of age, who suffered from internal carcinoma of the larynx. The left sided infiltration had already extended over the anterior commissure to about one-third of the right larynx. The symptoms were severe hoarseness and a moderate feeling of stenosis in the larynx. I intended to follow Gluck's procedure strictly, but during the chloroform narcosis symptoms of suffocation suddenly set in, which induced me to alter the operation somewhat. After the T-shaped epidermis cut in front of the larynx had been made I immediately amputated the latter below the ring cartilage by a cut slanting upwards from front to back, after severing the thyroid cartilage from its surroundings, applying a ligature at its isthmus in two places, cutting through and freeing the trachea where the resection was to take place with two raspatories from both sides and from the esophagus. Then followed Gluck's sewing of the lower tracheal end into the skin, in order to reestablish the breathing under nar-

cosis with less danger and to completely prevent blood or mucus from entering the trachea. Then the larynx was freed from below upwards near to the upper rim of the ring cartilage from the esophagus to the frontal hypopharyngeal wall, partly by blunt, partly by sharp dissection, and the cornu superius of the cartilage was cut off on both sides with scissors. Until then the pharynx had not yet been opened. After the whole wound, especially the part of the hypopharynx and the esophagus and all exposed muscles had been covered with thick gauze strips the pharynx was opened subhyoidally and immediately tamponed with gauze to prevent flow of secretions and infection of the wound surface. Then followed the severing of the larynx from the pharynx and the sewing up between the root of the tongue and the upper stump of the front wall of the esophagus or hypopharynx. Finally, after making counter openings on both sides of the tracheal opening, the outer subcutaneous wound was thoroughly tamponed with iodoform gauze and the epidermis ends were sewn together. For one week only liquid food was given by means of a permanent esophagus catheter. The convalescence was excellent, all sutures healed per primam intentionem. After ten days food could be taken without the catheter and after two months the patient could be discharged cured. He was subsequently demonstrated at the monthly meeting of the Japanese Oto-rhino-laryngological Society together with the removed parts. The man is still living and, considering his age, 70 years, quite well and active. It should be added that he can speak in whispers and make himself understood, in the beginning with the aid of Gluck's rubber tube, but later without it. Encouraged by this success I have up to December of last year (three cases in this year cannot be counted in yet) performed the same operation in 106 cases of various forms of laryngeal carcinoma, both internal and external forms, with and without accompanying glandular metastasis on the neck, with the following results:

Thirty-six cases showed no recurrence after three years, and fifty-five cases relatively cured, including all successful cases within the last three years and those who were discharged cured but have been lost sight of. Ten cases recurred, two of them locally near the epiglottis, one at the thyroid,

which latter was completely cured after reoperation; in seven glandular returns appeared, some of which could be relatively cured by reoperation.

Six cases ended fatally within one to four weeks after the operation; one on the tenth day through erysipelas, another on the seventh day through uremic anuria; one case ended on the twenty-fifth day on account of general debility and three with infectious pneumonia. The infection was, however, not caused by the influx of blood into the trachea, but later (two cases within ten days, one after two weeks) on account of local suppuration of the wound.

Compared with Gluck's, my results do not appear so good, but I must mention that I have ventured to operate on cases too which, according to Cerny and others, were until now considered inoperable, on aged persons of 70 years and more, on patients who already showed glandular swellings on one or both sides of the neck, in the hope of reducing the number of inoperable cases as much as possible. Taking all this into consideration, I think I can be satisfied with my results. Moreover, the causes of death in these six cases were not the effects of the operation itself, but mostly some accidental neglect, which with greater watchfulness on the part of assistants and nurses may be avoided. I therefore feel myself justified in recommending the above method to you, adding the following critical remarks:

The modus operandi adopted by me is really not new. From remarks made by Gluck I know that a French specialist, Lubec, long before Gluck, performed a very similar operation, by resecting the trachea in the first stage and sewing its lower end into the skin, letting the principal operation follow a fortnight later. Here in America James Bell is said to have reported in 1909 eight successful cases, which he had operated on in a manner very similar to mine, saying: "Without previous tracheotomy the larynx is removed from below upwards, after sewing the trachea to the skin." It is really not my intention to raise here the question of priority. I only wish to state that as early as 1903 I used this method on a patient, on whom I intended to perform Gluck's total extirpation, when some difficulty accidentally arose on account of the patient, and that since then I have practiced this method for nineteen years in

more than a hundred cases of laryngeal carcinoma, finding that it is easier to perform and less risky, and that it provides the same protection against infectious pneumonia as Gluck's method. If it should be objected by Gluck that the procedure of laryngectomy—that is, the total extirpation of the larynx from below upwards—increases the risk of infection on account of the early opening of the trachea, I would say on the contrary that the earlier opening of the pharynx cavity, which is the essential part of his procedure, is a source of danger from wound infection. For even if the mouth and pharynx cavity are at once tamponed with long strips of gauze and the wound surface likewise packed with gauze, very often quantities of mouth or pharynx secretions exude from the cut with reflex retching or swallowing movements. This not only greatly hinders smooth working but can frequently give opportunity for infection. In this respect I think my way to be even better, because the opening of the pharynx cavity comes last, after the trachea and the larynx have been completely separated from their surroundings and all wound surfaces have been thoroughly tamponed. For a very short time only the pharynx cut remains open, for immediately after tamponing the mouth and pharynx cavity with gauze or sponges sutures are made between the base of the tongue and the upper rim of the anterior hypopharynx. Moreover, the early resection of the trachea and the sewing of its lower stump to the epidermis I consider to be not at all so dangerous as Gluck believed, because the breathing opening is thus far removed from the chief field of operation. The following points I carefully heeded during the operation:

(1) The operation must be made under the strictest aseptic and general surgical rules.

(2) It may often be performed under local anesthesia, as nearly all Austrian, French, Italian and other specialists have expressly recommended. I myself have used successfully in six cases novocain infiltration combined with previous injection of pantopon scopolamin. But considering that with such a long and complicated operation a general chloroform narcosis is much more convenient, because the patient very often still feels pain after simple anesthesia, and that the injection of pantopon scopolamin is not always harmless, as P. Heyman

has asserted, I have mostly used general chloroform narcosis and have never had any bad effects.

Gluck formerly recommended the formation of a large winged lap in front of the larynx for initial skin incisions, and later a median longitudinal cut from the level of the hyoid bone to the jugulum and two horizontal cuts at the level of the hyoid bone at the lower edge of the ring cartilage from the forepart of one sternocleidomastoideus to the other, as being best for opening up the field of operation in all its parts and to make a reliable drainage of the wound cavity possible after the operation. I prefer the T-shaped cut between the hyoid bone and the fossa jugularis, by which I could sufficiently fulfill Gluck's two conditions, and which in some respects seems to me even of greater advantage, inasmuch as the tracheal stump sewed in need not necessarily be freed from its surroundings but can remain in firm connection with it.

(4) In resecting the larynx any tearing or injury to the surrounding muscles, fasciæ, vesicular sheaths, etc., must be avoided as much as possible.

(5) The thyroid artery, if still quite healthy, is better left in by ligating it in the middle of the isthmus in two places and cutting through between. If, however, the carcinoma has already penetrated into the neighborhood of the gland and there is any suspicion of its being affected, it may likewise be removed. I have not been able to confirm that the removal of this gland is in any way harmful to the organism.

(6) As Gluck has already fully explained, I too advise the removal of the regional lymphatic glands and ducts Only in cases of well localized intrinsic carcinoma without glandular swellings I leave these parts alone. But in cases where the lymphatic glands already show swelling, or in extrinsic cancer I carefully try to remove all existing glands, even the smallest, together with all lymphatic vessels lying between the diseased part and the muscles and fasciæ. Cases where the metastatic glands are already of considerable size and adhere to the large vessels of the neck must be declared inoperable.

(7) I will not enter into details about the after-treatment, but make only some few remarks:

(a) The gauze strips left in the wound cavity are removed after one week if nothing untoward has occurred until then, and replaced by small drainage tubes.

(b) For feeding I frequently introduce the small permanent catheter through the nose, or in some cases two or three times a day the usual esophagus catheter orally into the stomach. But I prefer the former, as there is danger of piercing the pharyngeal sutures with the latter.

(c) If the healing process progresses well, the permanent catheter is removed after one week and the patient can be fed in the ordinary way. If the pharyngeal sutures have opened, however, the catheter must be left in longer.

(d) If the strength and the state of the patient permit it, it is advisable to make him get up as soon as possible and even let him walk about the room.

GENERAL REMARKS AND CONCLUSIONS.

From all the facts presented here we can clearly see that we are in the period of achievement as regards the treatment of laryngeal cancer, and that Virchow's words have been realized: "If cancer is, in its beginning and for some time longer, a local affection, it must be possible to cure it locally at this stage."

And there is no doubt that the earlier the beginning carcinoma of the larynx is diagnosed and treated by a suitable operation, the more likely the success of the operation, as Fränkel and others have repeatedly said. We also know that the prognosis of laryngeal cancer has, with the progress of laryngeal diagnosis and with the development of suitable operating methods, going hand in hand with the general progress of surgery, become greatly improved, as recent statistics show. But if we compare the number of cases which have been treated successfully, either by operation or otherwise, with those not operable or operated on unsuccessfully, it becomes clear that we must not rest satisfied with what we have achieved. In our university clinics, where roughly 8,000 patients annually are taken in, and in my private hospital, I have seen within these last 20 years over 400 cases of laryngeal cancer, of which the majority for various reasons could not be operated on, partly on account of extensive glandular metastases, partly because of severe cachexia or general de-

bility, or on account of coexistence of other diseases, affections of the kidneys, heart, lungs, etc., and often also because of the refusal of an operation by the patient or his relatives. Our aim must be to improve the prognosis. To this end I wish to express our desiderata in the following concluding sentences:

(1) We must instruct the general public and practicing physicians by writing and by speech, that laryngeal cancer may begin without a known cause and without severe symptoms, sometimes with simple hoarseness or with slight stinging pains in the throat, symptoms which may probably be diagnosed by the ordinary physician as nothing more than laryngitis simplex, and that just this beginning form, prognostically and therapeutically, is most important, for it is certain that this form can with great probability be cured by a comparatively simple and harmless endolaryngeal operation or by thyrotomy.

(2) Practicing physicians, family and companies' doctors often have opportunities of observing these beginning forms and should become at least as familiar with laryngoscopic as with stethoscopic examinations. To attain this end I would propose to improve the methods of teaching laryngoscopy and to raise the position of laryngology in the realm of medical science.

(3) We specialists should in all suspicious cases endeavor to make an exact clinical diagnosis as early as possible, combined with microscopic examination of a test piece which has been immediately cut out after the laryngoscopic examination. If exact diagnosis shows the case to be one of cancer, a suitable method of operation must be selected according to the exactly ascertained nature of the case.

(4) We must not mechanically stick to only one method of operation, but must be able to individualize and prefer the smaller operation to the larger; but in case the indications should point to the necessity of the larger operation, we must not hesitate in employing the same.

(5) For selecting a suitable method I would propose my modus, which has proved practicable by long experience: Sharply defined polypoid forms on the free edge of the vocal cord, which have not yet penetrated into the ventricular membrane, or the movable forms of the pedunculated carcinoma are best operated on by the endolaryngeal excision of the

tumor, of course by a well practiced hand, with indirect method or with the suspension laryngoscope. In case these localized forms of the laryngeal cancer have already penetrated deeper into the ventricle, or in cases of primary ventricular forms (mostly adenocarcinoma), or if the surgeon is in doubt as to his dexterity for an endolaryngeal operation, I should recommend thyrotomy with subperichonderal excision of the tumor afterwards.

In cases where the carcinoma has not only affected the vocal cords but also the arytenoid cartilage or the cricoarytenoid articulation, or the posterior wall of the larynx, or has progressed over the anterior commissure to the other side, total extirpation must immediately be resorted to, if the constitution of the patient permits.

Partial resection of the larynx and hemilaryngectomy are, according to my experience, not very practicable; they may even prove dangerous, as it is difficult to separate the wound cavity from the air passage by a tissue wall. I would recommend this method only for sharply defined epiglottis carcinoma, as we can insure security against infectious pneumonia by a temporary closure of the introitus larynx by the suturing of the base of the tongue to the upper rim of the posterior wall of the larynx. Hemilaryngectomy would be better replaced by total extirpation.

This brings my short exposition to a close. Gentlemen, I thank you for the patience with which you have listened to my inadequate remarks on the subject.

XXXIX

THE STUDY OF THE TONAL RANGES IN LESIONS OF THE MIDDLE EAR.

By L. W. Dean, M. D., and C. C. Bunch, Ph. D.,

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The research work which we are reporting represents a large amount of careful, conscientious work, especially on the part of Dr. Bunch, who either made or supervised all the functional tests of hearing which are indicated. This report is confined to the examination of the auditory perception of a large series of cases of so-called middle ear trouble. In all cases exhaustive clinical examination was made. These examinations were grouped and studied, and the results are here summarized and presented for your consideration.

Plate 1 is the diagram of the clinical record made for each case examined. In only a few cases where it seemed desirable to have specific information regarding the vestibular functions were the turning tests made.

The hearing for whispered and spoken voice was first determined. Next the pitch range audiometer was used to determine the audition for tones between 30 and 7070 d. v.

The records with the audiometer are easily indicated by means of a graph shown in the plate. The pitch of the tones is indicated at the bottom and the loudness of the tone by the height of the curve on the graph, no tones at all being normally heard at zero. The curve shown on the graph is the field of hearing for a normal ear.

In the curves of cases studied that we are going to show, the curve was secured by testing the audition of the ear examined for every tone from 30 d. v. to 7070 d. v. The curve thus secured compared with the normal curve, which is stamped on every chart, gives an approximate idea of the relative hearing power for these tones.

If, for instance, the curve at 1,000 d. v. is only half as high as normal, this does not mean that hearing for that note

is one-half normal but only that hearing for the note is markedly decreased. We are not justified in assuming that the curves represent the exact audition for the note that is being tested. The fractions which express quantitatively the audition for various notes, when this is determined with the Bezold forks, are likewise very inexact. As the intensity of sound varies with the square of the distance, with the square of the amplitude of the vibration, and is influenced by the rapidity of vibration, it is an extremely difficult matter to determine of vibration of sound, it is an extremely difficult matter to determine in an yway the exact amount of audition for any given note.

If the curve of the ear being examined should show a tone gap in the tonal range, this does not necessarily mean that there is an absolute tone gap but it does indicate that the ear is unable to perceive the loudest tones capable of being produced by the audiometer. These gaps found by the audiometer have been verified in the psychologic laboratories by tests with organ pipes and other instruments producing pure loud tones. It is easy to conceive that if a note could be produced of greater intensity than that which the audiometer can give, it might be heard when the audiometer would produce no sensation of sound.

In the following charts the normal curve is indicated by a continuous line; curves for the right ear by dashes; and for the left ear by dots.

For the benefit of those who are not interested in the audiometer readings we have made in each case a quantitative determination of the audition for the 50 d. v. fork, the c, c^1, c^2, c^3, c^4, and c^5 forks.

The Rinne, the Weber, and the bone conduction tests were made with the 256 fork. In some cases the 128 and 512 d. v. fork was also used. These forks were weighted to minimize the production of confusing overtones.

At the bottom of the plate next to the audiometer curve is a measurement of the upper tonal limit, as determined with the Galton whistle, the Koenig cylinders and the monochord. The upper limit of the monochord is determined both by A. C. and by B. C. With the Galton whistle which we use the normal upper limit is 21,000 d. v. With the Koenig cylinders the

upper limit is 43,960 single vibrations and with the monochord by A. C. 19,000 d. v., by B. C. 20,000 d. v.

In each case studied we have been interested in determining if in addition to the middle ear lesion there was also present a lesion of nerve or end organ.

Suggestive of a lesion of the acoustic nerve or its end organ is the following:

1. The audition for the higher notes is poorer than that for the lower or middle part of the tonal range. If the lower notes of the tonal range up to about 3,000 d. v. are heard normally and there is a diminution in the audition for the notes in the upper part of the range, as is shown by Plate 2, this diminution of audition for the higher notes is suggestive of a lesion of the nerve or end organ. If, however, as is shown in Plate 3, there is a uniform diminution in audition for tones throughout the range, it does not indicate a lesion of nerve or end organ. The curves shown in Plate 3 are the curves secured from: first, a case of impacted wax in the external canal; second, a similar case; third, three cases of individuals with normal hearing where the external canal has been packed with boric acid powder, and lastly, a case of cotton in the ear which was soaked with cerumen and was in contact with the drumhead. In each of these cases you note the diminution in audition for all tones throughout the range. As we view it, there would have to be a distinctly greater diminution for the notes in the upper part of the range as compared with the tone best heard in the lower or middle part in order to indicate positively a lesion of the nerve or end organ. In some of these cases the c^5 fork was not heard at all.

Plate 4 shows the curves of these cases after the removal of the impacted wax, cotton or boric acid powder. Note that the curves are practically normal.

2. A lowering of the upper tonal limit, especially by B. C. The better the hearing for voice and whisper, the more significant of a lesion of nerve or end organ is the decrease in the upper limit by A. C.

Plate 5 indicates the upper tonal limit in three of the cases of obstruction in the external canal just described by A. C. and B. C. determined with the monochord. Note the upper limit by A. C. is lowered 2,000 d. v. from obstruction in the

external canal. There is, however, no cutting down of the upper limit by B. C., and we feel that a lowering of the upper limit by bone conduction is a more definite indication of a lesion of nerve or end organ than the lowering of the upper limit by A. C.

Plate 6. The comparison of the upper limit of audition for high notes as determined by A. C. and B. C. is of interest.

The upper chart shows the A. C. and B. C. as determined with the monochord for twenty-two acute suppurative ears. The vertical figures represent the number of cases; the horizontal the upper limit in double vibrations per second. The curves are similar, but by air the lowest limit was 12,000 d. v., while by B. C. it was 14,000.

The results of examination of twenty-eight cases of chronic otorrhea are charted in the center. Again the larger number of cases make similar curves, but here, while the lowest limit by B. C. is 13,000 d. v., by A. C. it is 6,000—a great difference.

Below are graphs secured for charting the results of forty cases of hyperplastic otitis media. Here the similarity of the curves is lost.

The diagnostic significance of the difference between the upper limit as determined by bone and air conduction with the monochord is here illustrated. In cases of acute trouble 22 per cent gave a difference of 23,000 d. v. or more between the upper limits determined by air conduction and by bone conduction. In the chronic otorrhea cases this percentage was 39, while cases of hyperplastic otitis media gave this difference in 67 per cent.

The determination of the upper limit by B. C. is a far more accurate indication of the condition of the nerve and end organ than its determination by A. C.

3. Positive Rinne.

4. The sensitivity for one note or a group of notes is much less acute than the others when determined with the tuning fork or the audiometer.

To determine whether there is a significant difference between the hearing power for high tones and low tones, we have used the audiometer and the Bezold forks. For example, if the hearing for the c^2 fork is indicated by the fraction 20/24 while that for the c^5 fork is indicated by the fraction 5/60,

there is a greater decrease for the c^5 than for the c^2. This would be shown on the audiometer record by a curve which closely follows the normal curve for a frequency of 512 d. v., but which falls much below the normal curve at 4096 d. v.

Plate 7 is a plate of neurolabyrinthitis showing these distinct depressions in the audiometer curve confirmed by the findings with the tuning forks.

5. Decreased bone conduction as determined by the c^1 and c^2 fork.

This is a very important matter. We feel that a bone conduction decreased two or three seconds is suggestive of a lesion of nerve or end organ when the examination had been properly made. We test bone conduction by placing the tuning fork over the bone just adjacent to the external opening of the bony canal posterior to the upper attachment of the external ear. We keep in mind the influence of mastoid involvement decreasing the bone conduction.

Bezold (Textbook of Otology, page 67) thinks that to be significant the perception by bone conduction for the fork placed on the vortex should be very much shortened.

Wittmaack (Zeit. f. Ohr., vol. 60, page 136) is of the opinion that a shortening of the bone conduction is worthy of diagnosis when it amounts to at least 30 per cent of the average value which we get or is received through the examination of a large number of normally hearing individuals with the chosen tuning fork.

As the percentage of decrease will depend altogether upon the energy with which the fork is excited, this statement is of little value.

Bezold feels that an objection to this procedure is that extraneous sounds and the ability of the individual to make an accurate examination and of the patient to cooperate have a decided bearing upon the result. Such criticism is valid for all functional tests of hearing and is as true for determining hearing power by A. C. as well as B. C.

Our examinations are conducted in a room set apart especially for this purpose and, while it cannot be said to be entirely free from external noises, it is probably much more nearly ideal for this purpose than those ordinarily used.

Emerson (ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Dec. 21, page 999) is of the opinion when B. C. is lowered for the 256 or 512 d. v., that fork nerve deafness has been present for a long time.

Werhofsky (Prufung. d. Herdauer in Verlaufe der Tonskala bei Erk. d. Mit. u. Innern O. Bezold U. d. Funk, Pruf. d. Mench. Gehor., 1897-1909) found decreased B. C. in all lesions of the nerve or end organ that he studied. He reports B. C. decreased two seconds in one case of nerve deafness and three seconds in another.

He says that B. C. is less with higher forks than with lower and cites a number of observations to confirm this.

In describing his technic, he does not state the length of time his forks vibrate with a given stimulus. Our higher forks vibrate longer than the lower, and consequently the decrease in B. C. with the higher forks would be greater.

Siebenmann in an article translated by Dench (Archives of Otology, vol. 22, p. 5) says that in apparently normal ears there may be functional defects in the test for the upper tone limit and in the perception of sound by B. C. He also shows that B. C. is sometimes decreased by aspiration of the middle ear.

Page (Proceedings N. Y. Acad. of Med., Oct. 13, 1921) says that the early cases of otitis media have a negative pressure in the tympanum.

Kerrison (Text Book of Otology, pg. 89) suggests that the hearing by bone should be decidedly decreased to be significant.

Schwabach (Uber Harprufung, etc., Archiv. f. Ohren., vol. 31, page 6) says that B. C. is longer than with normal persons in lesions of the transmitting apparatus, while it is the same as normal persons or less in lesions of the perceiving apparatus of the ear.

In our study of acute middle ear infections we have found it very unusual to find an infected ear without evidence of a lesion of its nerve or end organ.

Siebenmann, in an article translated by Dench (Archives of Otology, vol. 22, page 12) reports that in making functional examinations of pure cases of catarrh of the eustachian tubes he found the upper tone limit usually lowered and not restored by inflation. He assumed that the lowering of the upper limit

was due to a passive hyperemia of that portion of the scala vestibuli lying next the oval window.

Plate 8 shows curves of a case of acute tubal catarrh where we found no definite evidence of lesion of nerve or end organ. The decrease in hearing for the voice and whisper was apparently due to the middle ear condition and disappeared wholly on inflation. B. C. in each ear was normal. Upper limit by monochord, R., A. C. 16,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000. As with the blocking of the external canal the upper limit was lowered by air and not by B. C. Unfortunately, after inflation the upper limit was not tested by A. C.

The curve before inflation shows rather a uniform decrease in audition for all notes except the very lowest, which were not heard at all. The audition for the notes became almost normal on inflation.

Twenty-two acute infected ears were studied. Of the twenty-two, fourteen had positive Rinne and eight had negative Rinne; fifteen had decreased bone conduction for the c^2 fork and fourteen had the upper tone limit decreased 2,000 or more by A. C. as determined by the monochord. Thirteen of the fourteen had the upper limit by B. C. decreased 2,000 or more. Twelve cases had decreased bone conduction as well as lowered upper limit. Two cases had decreased B. C. with upper limit normal and two cases had upper limit by A. C. lowered but B. C. normal.

Plate 9 shows the curves of ten acute infected ears. The dots, dashes and circles in the curves are added so that they may be more easily followed through the entire field. You will note that curves Nos. 2 and 3 show complete gaps. Partial gaps are shown in all the curves except No. 4. Ninety per cent of the curves of acute infected ears are suggestive in this manner of lesions of the nerve or end organ.

A careful analysis of the clinical charts of the twenty-two ears studied shows that in every acute infected ear except one there was either decreased B. C., upper limit lowered by B. C. 2,000 or more, or a defect in the audiometer curve suggestive of lesion of nerve or end organ.

These evidences of lesion of nerve or end organ appear very early in the course of the disease.

Plate 10. H. B. Examination was made within twelve hours after first evidence of infection. With good hearing for voice, spoken, 45 feet, and whisper, 15 feet, in each ear; frank earache in the left, slight earache in the right; drumheads reddened, no bulging; Rinne positive in each ear, c^5 fork in left 30/60; with other forks normal. Two partial tone gaps in left ear with audiometer curve. B. C. 2 sec. each; upper limit lowered 2,000 by B. C. with monochord. No leucocytosis at this time. Patient discharged ten days later, B. C. normal; c^5 heard normally, upper limit normal by A. C. and B. C.

In cases where the infection was unilateral the evidences of a lesion of nerve or end organ were not confined to the infected ear.

Plate 11 shows the findings of the good ear in six cases of unilateral acute infection of the middle ear. Only one case, the first, had no suggestion lesion of end organ or nerve in the good ear. In the second, third, fourth and fifth cases the upper limit is lowered; in the fourth, fifth and sixth B. C. is decreased. The audiometer curves of the first case, and of cases three and four, were somewhat suggestive of lesion of nerve or end organ so that in at least five out of six of the unaffected ears, and probably all six, there is evidence of involvement of the nerve or end organ.

Plate 12 B. D. This case is interesting because a few days before infection of the left ear the ears were tested and were apparently normal. Note that in the right ear where there was no redness of drumhead or other evidence of middle ear infection, the decreased hearing for voice and whisper, the decreased B. C., the decreased upper limit by B. C., and the slight diminution for the c^1, c^2, c^3, c^4 and c^5 forks, for the left ear, the decreased B. C., the tuning fork tests and the audiometer curve are all suggestive of lesion of nerve or end organ.

Treatment and eradication of the middle ear condition may have no influence upon the lesion of nerve or end organ.

Plate 13 E. B. shows the findings at the height of an acute tubal catarrh. The tubal orifices were edematous, covered with dilated blood vessels; the drumheads were reddened, not bulging. B. C. decreased each 2 secs, Upper limit each B. C. 17,000 by monochord.

Plate 14 shows the findings after the eradication of the middle ear lesion. B. C. —3. Upper limit lowered by B. C. Audiometer curve indicative of lesion of nerve or end organ. The findings remained the same as long as the patient remained under observation.

Plate 15 shows the findings A. A. S., taken one month after an infection of the left ear was apparently well. Curve and tuning fork findings for left ear indicate a lesion of importance involving nerve and end organ. Whisper and voice are poorly heard.

Plate 16 B. D. shows findings in both ears in a case of acute infection of the left ear one month after discharged as cured. The curve left, tuning fork tests left, and decreased B. C. each indicates a lesion of nerve or end organ left and probably also right. At no time during the disease was there evidence of acute middle ear infection right. Nevertheless, the hearing right which was W15, V45, before the infection of the left ear is now W13, S45, while the B. C. is —5. Before the infection of the left ear it was normal.

After acute infections of the middle ear with return to normal audition for voice and whisper there may be definite indications of lesions of nerve or end organ.

Twelve cases of bilateral chronic otorrhea and fifteen cases of unilateral chronic otorrhea, or thirty-nine ears, suffering with chronic otorrhea, were studied. Rinne was negative in thirty-five ears, positive in four. There was decreased bone conduction in eleven ears, increased in twenty-six, normal in two. Decreased B. C. was not so common as in the acute infections. The upper tone limit was decreased 2,000 or more by bone conduction in thirty ears. In thirty-seven ears the audiometer curve was suggestive of a lesion of nerve or end organ and in two ears not. Marked depressions and tone gaps in the audiometer curves were much more frequent than in the acute ears. The curves were much more characteristic of lesions of the nerve or end organ. If we include the results with the tuning fork tests, in only one ear out of the thirty-nine was there no evidence of a lesion of the nerve or end organ.

Plate 17 R. B. is a typical chart showing the findings in a case of chronic otorrhea. Note that with the tuning forks the

hearing range is from c^1 to c^4 inclusive. The audiometer curve shows the decreased audition for both low and high notes in the range of the audiometer with depressions characteristic lesions of the nerve or end organ. The upper limit in each ear is decreased to 15,000 d. v. or B. C.

From a study of the cases of chronic otorrhea one gets the impression that the extent of the lesion of nerve or end organ corresponded roughly with that of the middle ear lesion, while in the acute cases there was the greatest variation between the extent of the lesions of the inner ear and those of the middle ear. It was not unusual with a very mild acute middle ear infection to have a very well marked lesion of the nerve or end organ. A study of the fifteen unaffected ears in the fifteen cases of unilateral chronic otorrhea shows that Rinne was negative in unaffected ears no times, positive in fifteen. B. C. was decreased in the unaffected ears three times; it was normal ten times and increased twice; the upper limit was decreased by B. C. 2,000 or more five times and was normal ten times. The audiometer curve of the unaffected ears was indicative of a lesion of nerve or end organ in only one case. The unaffected ears in this series of unilateral cases were very good ears with one exception. The hearing in almost every ear was W 15 feet. While these findings suggest that there are a fair number of lesions of the nerve or end organ in the unaffected ears in chronic otorrhea, we do not find the large percentage of involvement of the nerve or end organ as in the unaffected ears in the acute infections. This suggests to us that the involvement of the nerve or end organ in the unaffected ears in acute infections is frequently not a permanent affair; otherwise, we would find the same number of lesions in unaffected ears in chronic otorrhea. The lesions of the nerve or end organ in ears suffering from chronic otorrhea were much more marked than in the acute infections. There is a direct interdependence between the chronic otorrhea and the lesions of the nerve or end organ. The chronic otorrhea has no influence of any moment upon the nerve or end organ of the opposite side. In our acute cases we get the impression that the acute middle ear trouble and the lesion of the nerve and end organ are not interdependent. They may both be due to the same cause but not necessarily so.

Plate 18 is a composite graph of the results obtained from tuning fork examinations. It is difficult to express in a uniform manner the results obtained in tuning fork examinations when different denominators are used to represent normal hearing for different tuning forks. For example, in measuring the hearing power for the tuning forks we represent the hearing for a normal ear in terms of distance as the denominator of a fraction. The hearing power for that fork by a pathologic ear is recorded in the same terms in the numerator. For the c^5 fork, since a very slight impact produces a sound heard at a considerable distance, we use the denominator 60. The 256 fork is heard at a less distance and we use 24 for the denominator. For similar reasons we use the denominator 4 with the 50 d. v. fork.

In order to make an analysis of our results we have arbitrarily divided our results into six classes. Those who do not hear the fork at all are put in Class 1; those who hear the fork when it is very loud, as indicated by fractions equivalent to one-fourth or less, we have put in Class 2; those who hear it by values indicated by the fractions between one-fourth and one-half are put in Class 3; those who hear it by fractions from one-half to three-fourths are put in Class 4; those who hear it from three-fourths to less than normal are put in Class 5; and those who hear it normally are put in Class 6. The curve shows that twenty-one cases did not hear the 50 d. v. fork at all; three did not hear the c^5 fork; fourteen heard the 50 d. v. fork with a fraction equivalent to one-fourth or less; while twenty-seven heard the c^5 fork with results indicated by a fraction of one-fourth or less. You will notice that the 512 d. v., 1024 d. v. and 2048 d. v. forks run almost parallel throughout the entire curve, while the greatest variations in chronic otorrhea cases are shown with the 50 d. v. fork and the 4096 d. v. fork. There were no cases where the hearing was perfect for the low fork and two cases where the hearing was normal for the 4096 d. v. fork.

Plate 19 shows the results of the audiometer examinations in ten cases of chronic otorrhea. It is inserted to show the frequency with which such phenomena as tone islands, gaps and other peculiarities exist. These peculiarities are indicative, in our judgment, of lesions of the nerve or end organ.

For example, curve No. 8 shows simply a region of residual hearing for tones between 500 d. v. and 1500 d. v. No. 7, while rising much higher on the chart, shows two islands with the interlying gaps at 2500 d. v. and at about 3200 d. v. Curve No. 9, even though rising to the normal at 1000 d. v., shows that the low tones are lost, that there is a decrease for notes above 1200 d. v., and a return to almost normal at 5500 d. v.

Sixty-six per cent of the records of cases with chronic otorrhea show either complete or marked partial tone gaps. These do not include such cases as are illustrated by curves Nos. 1, 2 and 8, which are equally significant of an inner ear lesion.

In only one case, L. P., age 13, did a tone gap disappear with treatment.

Hyperplastic Otitis Media.—Thirty cases of bilateral H. O. M. and four cases of unilateral H. O. M., or sixty-four ears, were studied. Rinne was negative in thirty-three ears, positive in thirty ears, questionable in one ear. There was decreased bone conduction in forty-four ears, increased bone conduction in sixteen ears, normal in four ears. The upper limit was decreased by B. C. 2,000 or more d. v. in forty-nine ears.

Twenty-one ears had complete tone gaps in the audiometer findings. In eight more there were marked partial gaps or 45 per cent showing these puecularities. In addition to this there were 34 cases where there were marked depressions equally indicative of inner ear involvement.

Plate 20 shows the curves of ten typical cases of H. O. M. Complete tone gaps are illustrated in curve No. 10, which has a gap at 2400 d. v. and another at 2000. Curve No. 9 shows a dip or partial gap at 1600 d. v. No. 6, while not showing either a partial or complete gap, shows that the hearing was normal up to 1500 d. v. and decreased more and more for tones higher in the scale. A curve of this type certainly indicates a lesion of the nerve or end organ. Curve No. 8 shows only a patch of residual hearing between 100 d. v. and 1500 d. v. Each curve shows marked diminution in the audition for the c^5 fork. In the next plate you will note that in only eight of the sixty-four ears was the c^5 fork heard normally.

Plate 21 shows a composite of the tuning fork cases in hyperplastic otitis media. Here we have made the same arbi-

trary divisions as we did with Plate No. 18. Here again we see that the larger variation is shown with the 4096 d. v. and the 50 d. v. forks. The 1024 d. v. and the 2048 d. v. forks run almost parallel through the entire range. While there were no cases where the 4096 d. v. fork was not heard there were 51 cases who could hear this fork only when it was struck very loud as, for instance, when the fork was forcibly struck against the heel of the shoe, twenty cases did not hear the 50 d. v. fork.

Note that in eight cases the 50 d. v. fork was heard apparently normal. In these eight cases the lesion of the nerve or end organ overshadowed the middle ear lesion.

A study of the clinical charts of these cases shows evidence of the involvement of the nerve or end organ in every ear affected save one.

Plate 22 F. K. shows the curves of a bilateral case of H. O. M. Note the negative Rinne, slightly decreased B. C., power of hearing low and high notes markedly interfered with in each ear, tone gaps in the audiometer curve, and upper limit markedly decreased both by A. C. and B. C.

Plate 23 C. L. shows in the right ear bone conduction normal, and negative Rinne, audiometer curve practically normal, upper limit normal. In the left ear B. C. is decreased five seconds. The right ear of this case was the only one of the sixty-four ears studied that did not show some evidence of involvement of nerve or end organ. Hearing was poorer in the right ear.

There was found no definite relation between the extent of the inner ear lesion as compared with the middle ear lesion.

Plate 24 shows the findings in H. S., aged 62, and in C. P., aged 62. The objective findings of the middle ear were similar. In one case the bone conduction is —5 seconds and in the second case it is —12 seconds. In the first case the audiometer curve shows a tone gap in the left ear and in the second case there is none. In the audiometer curve the upper notes are heard much poorer in the first than in the second. In the first case the upper limit is so low that it cannot be detected using the Galton whistles or the monochord, either by A. C. or B. C., and with the Koenig cylinders it is only approximately half normal, 21,000 s. v. instead of 41,000, while

in the second case the upper limit is decreased, it is 16,000 by B. C. with the monochord. The lesion of the nerve or end organ is much more marked in the first than the second case. In the second case, the one with the least involvement of the nerve, had a positive Wassermann, the first one not.

Plate 25 (Findings of Miss D. and H. B.).

The age of the first is twenty-five years and the age of the second is twenty years. The hearing in the left ear is approximately the same for whisper. In the first case B. C. left is increased, the tuning fork test shows fair hearing for the c^4 and c^5 fork, the upper limit by B. C. is normal, the audiometer curves, while there is a gap in the right ear and depressions in the left, shows fair hearing for some of the notes in the higher part of the range. In the second case, however, B. C. for c^1 fork is —5, c^2 fork —6, hearing for the c^4 and c^5 fork is markedly decreased, the audiometer curves show a cutting down of the higher notes to 4,000, the upper limit by B. C. is 15,000. The evidence points to a much more marked lesion of the nerve or end organ in the second case than in the first.

In one case of H. O. M., while under observation acute suppurative otitis developed. With the diminution in hearing the B. C., which was plus 4 in the left ear, became decreased. With the disappearance of the acute otitis it became plus again.

Our cases of H. O. M. frequently tell of improvement in hearing that is satisfactory to them following one kind of treatment or another. Our tests have failed to show any actual improvement in any case unless the lesion of the nerve or end organ greatly overshadowed the middle ear lesion, and the improvement resulted from the treatment of the case for syphilis, or removal of tonsils, or what not.

Plate 26. H. J. is a case of H. O. M. where there was also a plus 4 Wassermann. Note the positive Rinne with the c^2 fork and the greatly decreased B. C. with the same fork and a negative Rinne with the c^1 fork, which is decreased only six seconds. This patient's deafness has rapidly appeared in the last six months. It has progressed too rapidly for H. O. M. The syphilitic lesion of the nerve or end organ is more responsible for the deafness.

Plate 27. J. C. is a case of H. O. M. with negative Wassermann and no evidence of syphilis, where the lesion of nerve or end organ greatly overshadowed the middle ear lesion. Note the Rinne plus, B. C. decreased 6 seconds and 4 seconds, the tuning forks suggestive of lesion of nerve or end organ. The upper limits by B. C. 12,000 d. v. and 13,000 d. v. The curve is also characteristic of lesion of nerve or end organ.

A study of the unilateral cases of H. O. M. gives some very interesting information. Four cases were studied. In the unaffected ear, negative Rinne in no cases. Decreased B. C. in two cases, upper limit decreased 2000 or more by B. C. in three cases. In all four of the unaffected ears there was some evidence of lesion of nerve or end organ if we include the findings with tuning forks and audiometer.

CONCLUSIONS.

1. The determination of the upper limit by B. C. is a far more accurate indication of the condition of the acoustic nerve and its end organ than its determination by A. C.

2. It is usual in acute middle ear infections, even in the early stages, to find evidences of involvement of nerve or end organ. Rarely with the present methods of diagnosis are we unable to find evidences of such involvement.

3. The lesion of nerve or end organ is not necessarily dependent upon the acute middle ear infection. There is no definite relation between the severity of the middle ear infection and the lesion of nerve or end organ. When the middle ear infection is unilateral the unaffecetd ear usually presents evidence of a lesion of nerve or end organ.

4. After the disappearance of the acute middle ear infection there may remain a permanent lesion of nerve or end organ.

5. In every case of chronic otorrhea except one there was evidence of a lesion of nerve or end organ. There is an interdependence between the middle ear lesion and that of the nerve or end organ.

6. In the H. O. M. cases every ear studied save one presented evidence of lesion of nerve or end organ.

7. In H. O. M. there is no constant relation between the extent of the lesion in the middle ear and the lesion of the nerve or end organ.

OUTLINE FOR NEUROLOGICAL, NERVE DEAFNESS AND LABYRINTHINE CASES.

NAME DATE

	Before inflation		After inflation	
Hearing (Right	W	S	W	S
(Left	W	S	W	S

Paracusis Willisii
Tinnitus
Dizziness
Staggering
Vomiting

Tests

Rinne (Right Weber Politzer Bone conduction (R
(Left (L
Fistula Test:
Gelle Test:

Hearing	50DV	C	C^1	C^2	C^3	C^4	C^5
Right							
Left							

Low limit High limit

Balance sense

Romberg-walking crack On one foot Hopping, eye shut

Tests of vestibular apparatus

Spontaneous

Past-pointing

Extra ocular muscle balance

Maddox Dist. Near

Bi. Bo. Bu. Bd.

Nystagmus Up Down Right Left

Caloric

Right Left

After Min. Sec. After Min. Sec.

Nature of nystagmus Nature of nystagmus

Amp. Amp.

Head back
Head forward
Past-pointing
Falling

Rotation

(Eyes closed during rotation)

Head 30 degrees forward

R to L turns in 20 seconds:

Nystagmus Duration

L to R turns in 20 seconds

Nystagmus Duration

R to L 10 turns in 10 seconds:

Pointing Falling

L to R 10 turns in 10 seconds

Pointing Falling

Head forward 120 degrees

R to L 10 turns in 20 seconds

Nystagmus Duration

L to R 10 turns in 20 seconds

Nystagmus Duration

R to L 10 turns in 10 seconds

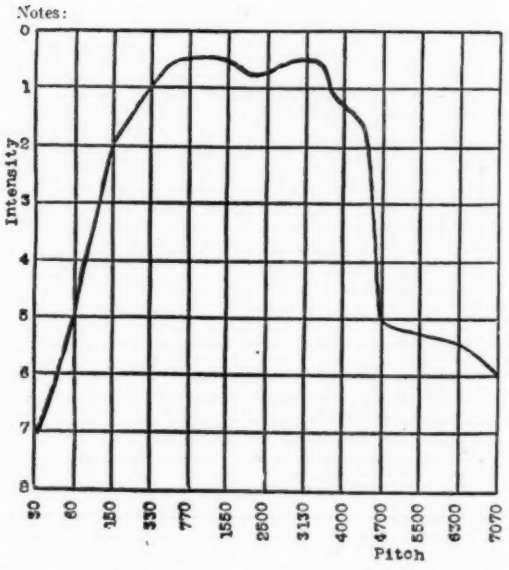
Pointing Falling

L to R 10 turns in 10 seconds

Pointing Falling

Vertigo during various tests:

PLATE I.



Upper limit: Galton, R., 23,000 d. v.; Koenig, R., 43,960 v. s.
Monochord: A. C., R., 19,000 d. v.; B. C., R., 20,000 d. v.

PLATE II.

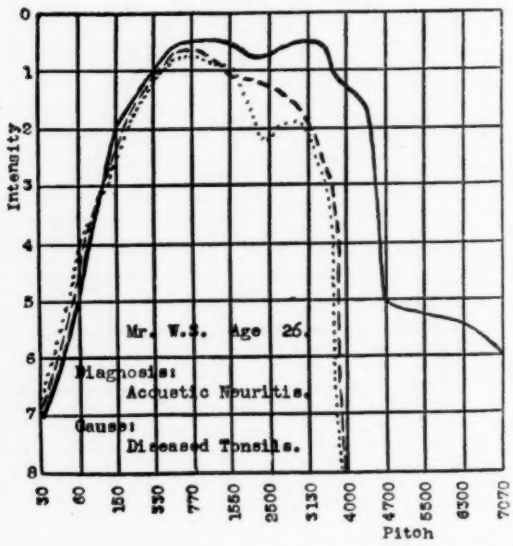

Intensity
0
1
2
3
4
5
6
7
8
Mr. W.S. Age 26.
Diagnosis:
Acoustic Neuritis.
Cause:
Diseased Tonsils.
30
60
150
330
770
1550
2500
3130
4000
4700
5500
6300
7070
Pitch

PLATE III.

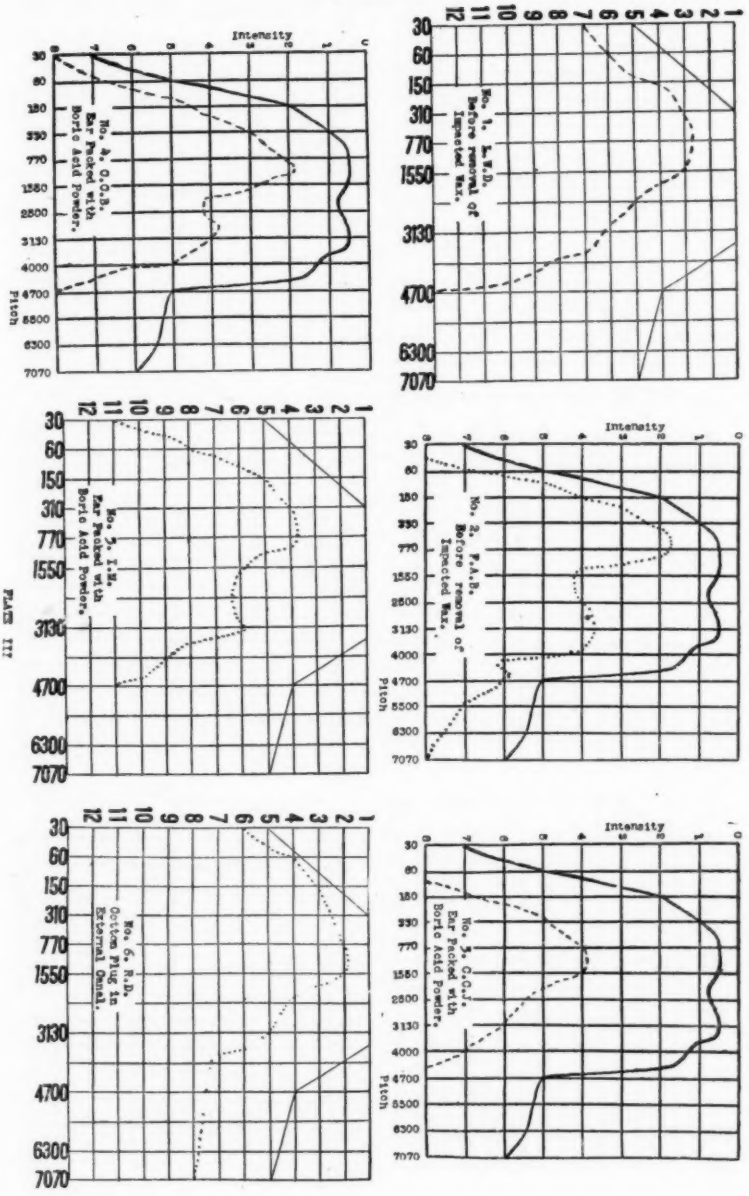


PLATE III

PLATE IV.

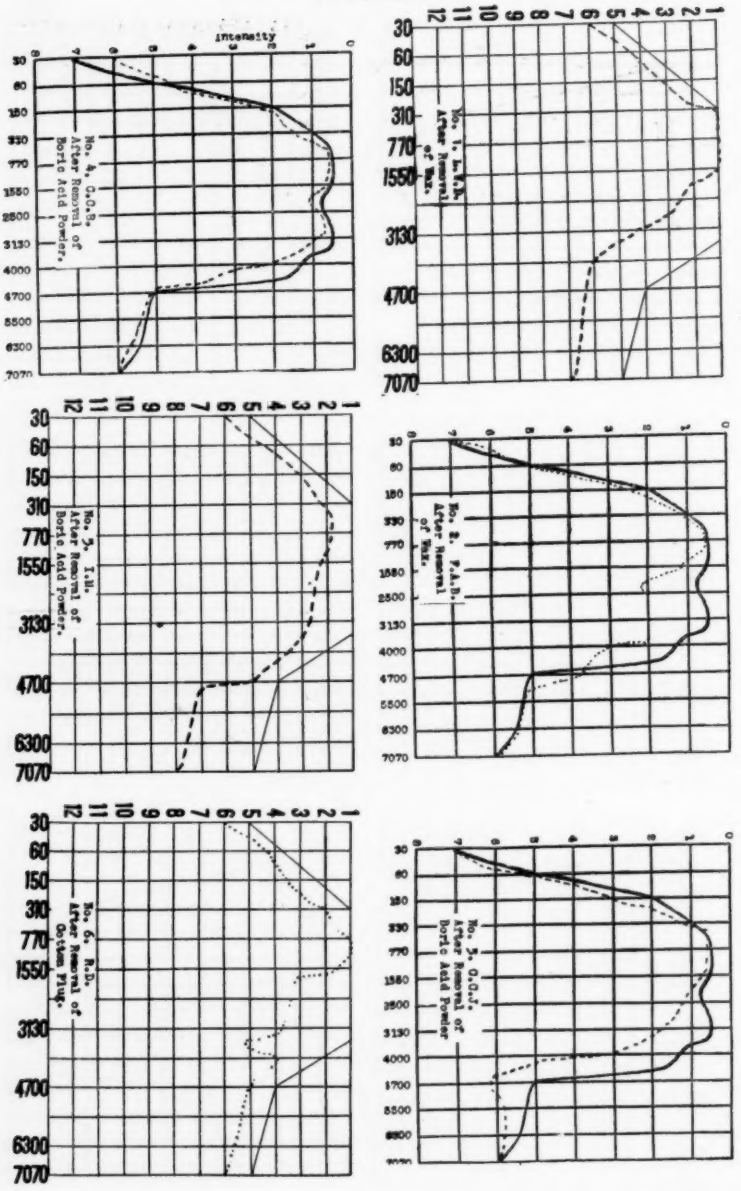

Intensity
No. 4, G.O.S.
After Removal of
Boric Acid Powder.
No. 1, L.W.D.
After Removal
of Wax.
No. 3, I.W.
After Removal of
Boric Acid Powder.
No. 2, F.A.D.
After Removal
of Wax.
No. 6, R.D.
After Removal of
Golden Plug.
No. 5, G.O.S.
After Removal of
Boric Acid Powder

PLATE V.

Upper limits in cases of occlusion of external auditory meatus.

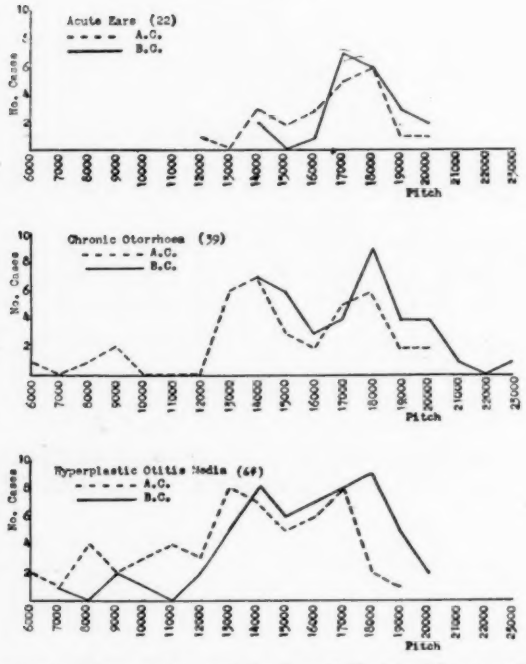
Before Removal

No. 2.	F. A. B.	A. C. 17,000	B. C. 19,000
No. 3.	C. C. J.	A. C. 15,000	B. C. 18,000
No. 4.	C. C. B.	A. C. 17,000	B. C. 20,000

After Removal.

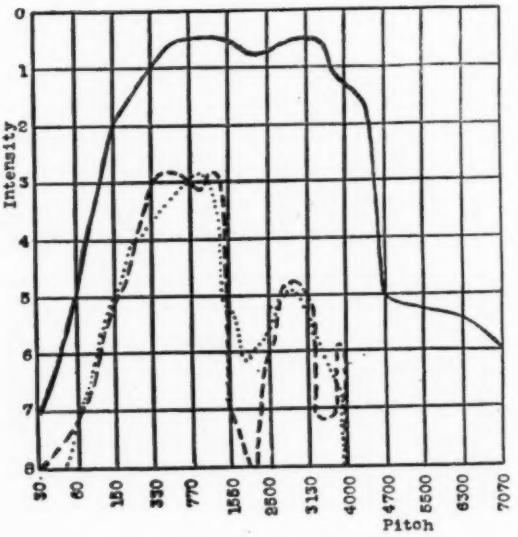
No. 2.	F. A. B.	A. C. 18,000	B. C. 19,000
No. 3.	C. C. J.	A. C. 17,000	B. C. 19,000
No. 4.	C. C. B.	A. C. 19,000	B. C. 20,000

PLATE VI.



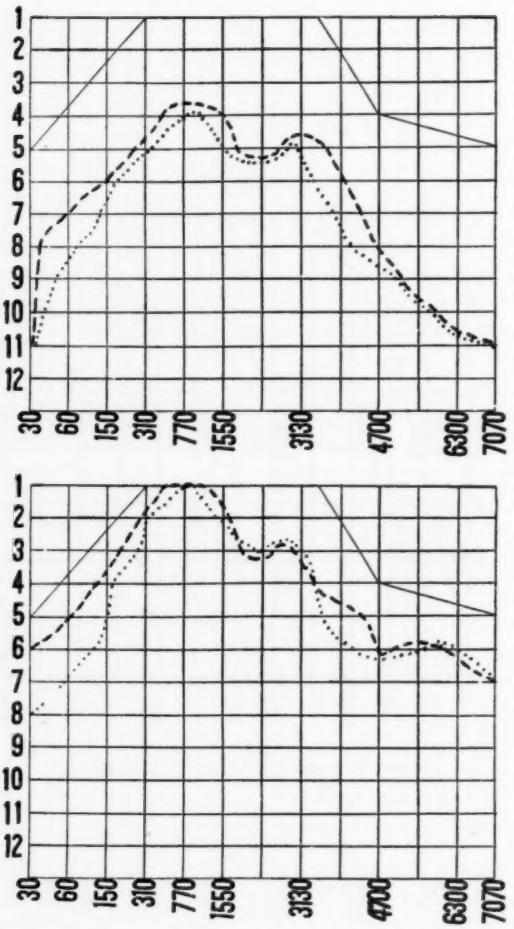
Upper limit—Comparisons of A. C. and B. C. with monochord.

PLATE VII.



Mr. N., age 17. Cause: Mumps.

PLATE VIII.



Mr. C. C., age 15. Diagnosis: Acute tubal catarrh. Before inflation: Voice, R., Wh. 9 ft., Sp. 16 ft.; L., Wh. 6 ft., Sp. 15 ft. After inflation: R., Wh. 15 ft., Sp. 45 ft.; L., Wh. 15 ft., Sp. 45 ft. Upper limit, monochord: R., A. C. 16,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000.

PLATE IX.

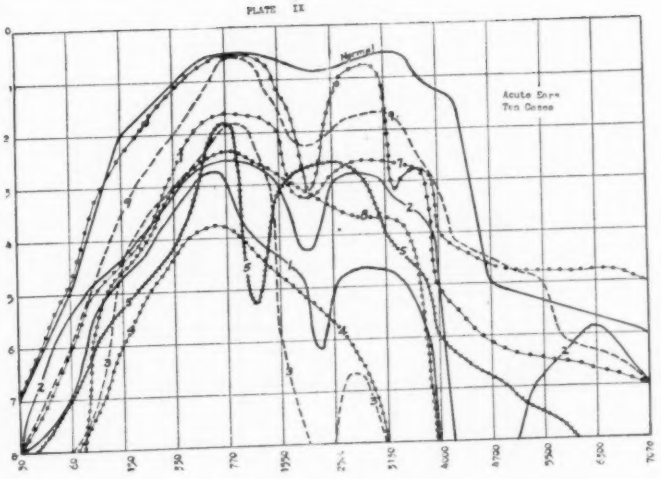
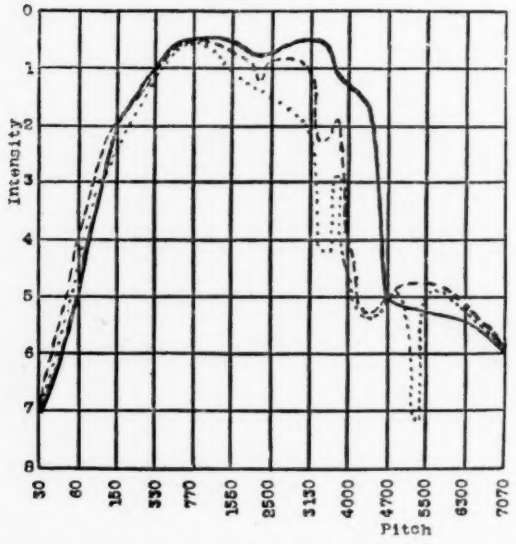


PLATE X.



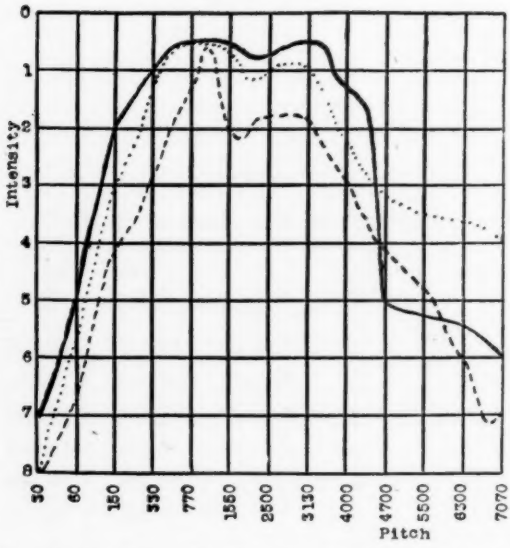
Mr. H. B., age 18. Diagnosis: Acute otitis media. (Twelve hours' duration.)

PLATE XI.

Uneffected Ears in Unilateral Cases.

Name	Rinne	B. C. (c_2 fork)	Monochord A. C.	Monochord B. C.
A. A. S.	+	Normal	19,000	19,000
M. K.	+	Normal	15,000	17,000
G. V. B.	+	Normal	16,000	17,000
E. C.	+	—	17,000	18,000
B. D.	+	—	18,000	18,000
E. S.	+	—	19,000	21,000

PLATE XII.

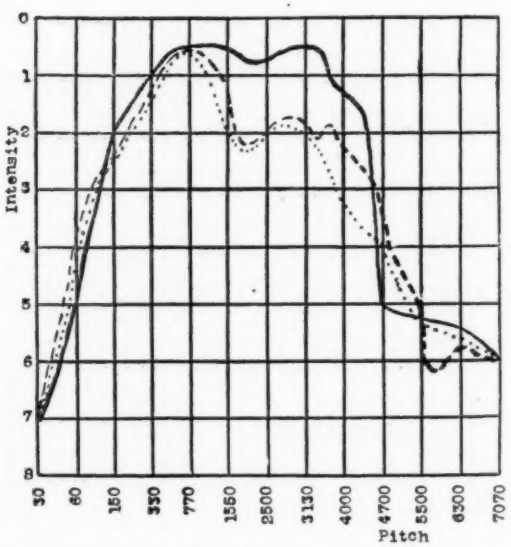


B. D., age 22. Voice, R., W. 8 ft., Sp. 40 ft.; L., W. 30 in., Sp. 36 ft. B. C. (c^2 fork), R., 1 sec.; L., 4 sec. Upper limit: Monochord—R., A. C. 18,000, B. C. 18,000; L., A. C. 18,000; B. C. 19,000.

Forks 50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R....	4/4	8/8	11/12	22/24	36/36	44/48	50/60
L....	4/4	8/8	10/12	20/24	36/36	40/48	40/60

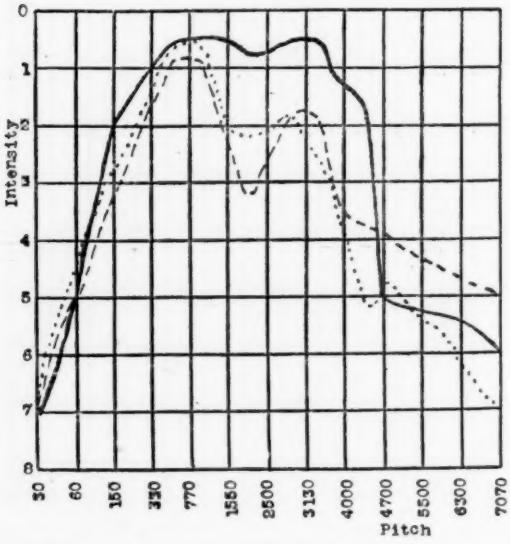
Diagnosis: Acute suppurative otitis media left.

PLATE XIII.



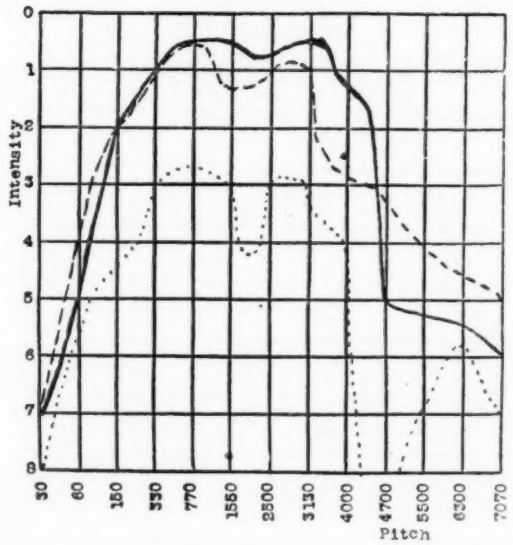
E. B., age 49. Voice: R., Wh. 12 ft., Sp. 45 ft.; L., Wh. 13 ft., Sp. 45 ft. B. C. (c^2 fork), R., —2 sec., L., —2 sec. Upper limit, monochord, R., A. C. 14,000, B. C. 17,000; L., A. C. 13,000, B. C. 17,000. Diagnosis: Acute tubal catarrh.

PLATE XIV.



E. B., age 49. B. C. (c^2 fork), R., —3 sec., L., —3 sec. Upper limit, monochord, R., A. C. 16,000, B. C. 17,000; L., A. C. 14,000; B. C., 17,000.

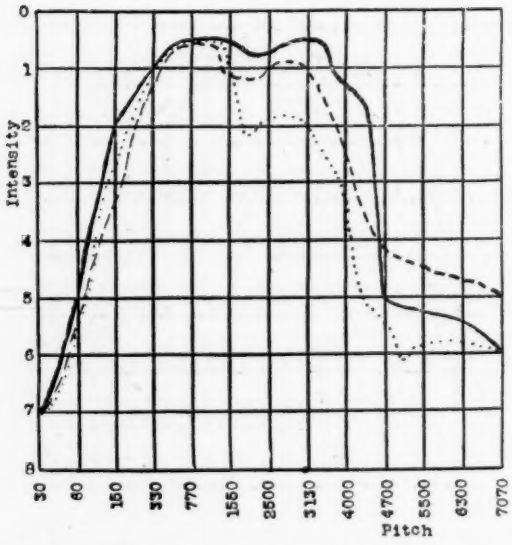
PLATE XV.



Mr. A. A. S., age 18. Voice, R., Wh. 15 ft., Sp. 45 ft.; L., Wh. 10 in., Sp. 2 ft.

Forks 50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R..... 4/4	8/8	12/12	24/24	36/36	48/48	55/60
L..... 3/4	6/8	7/12	18/24	24/36	20/48	15/60

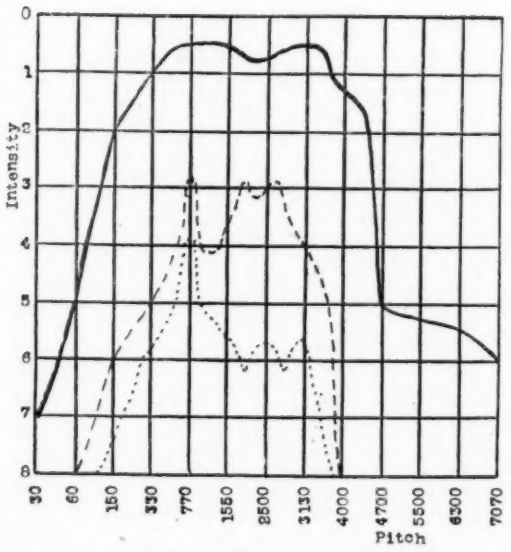
PLATE XVI.



Miss B. D., age 22. Voice, R., Wh. 13 ft., Sp. 45 ft.; L., Wh. 12 ft., Sp. 45 ft. B. C. (c^2 fork), R., —5 sec., L. —4 sec. Upper limit, monochord, R., A. C. 19,000, B. C. 19,000; L., A. C. 18,000, B. C. 19,000.

Forks 50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R.....	4/4	8/8	12/12	24/24	36/36	48/48	60/60
L.....	4/4	8/8	12/12	24/24	36/36	40/48	55/60

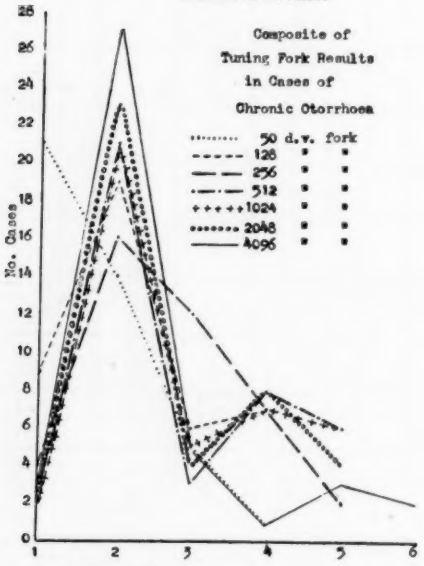
PLATE XVII.



R. B., age 14. Diagnosis: Bilateral chronic otorrhea, persisting after bilateral radical mastoid operation. Voice: R., Wh. 18 in., Sp. 3 ft.; L., Wh. 4 in., Sp. 9 in. B. C., R. and L. increased (c^2 fork). Rinne, R. and L. negative. Upper limit, monochord, R., A. C. 14,000, B. C. 15,000; L., A. C. 13,000, B. C. 15,000.

Forks 50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R.....	0	0/8	1/12	1/24	1/36	1/48	0/60
L.....	0	0/8	0/12	0/24	0/36	1/48	0/60

PLATE XVIII.



1 Not Heard
2 Heard by a fraction equivalent to or less than 1/4
3 " " " " between 1/4 and 1/2
4 " " " " " 1/2 " 3/4
5 " " " " " 3/4 " 1/1
6 Normal

PLATE XIX.

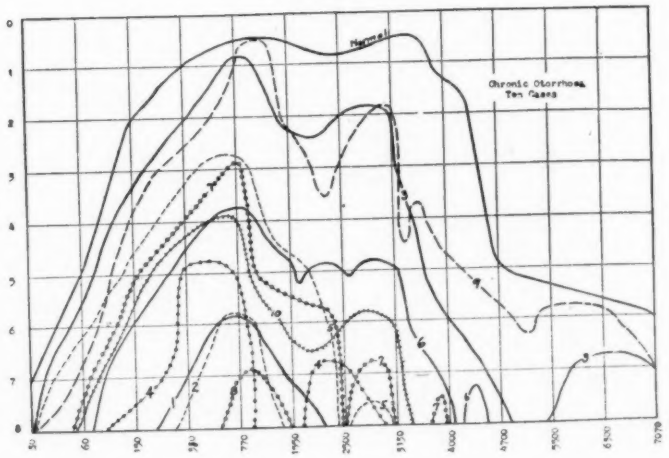

Normal
Chronic Otorrhoea
Ten Cases
0
1
2
3
4
5
6
7
8
50
60
190
580
770
1990
2900
5150
6000
6700
7900
8530
7079

PLATE XX.

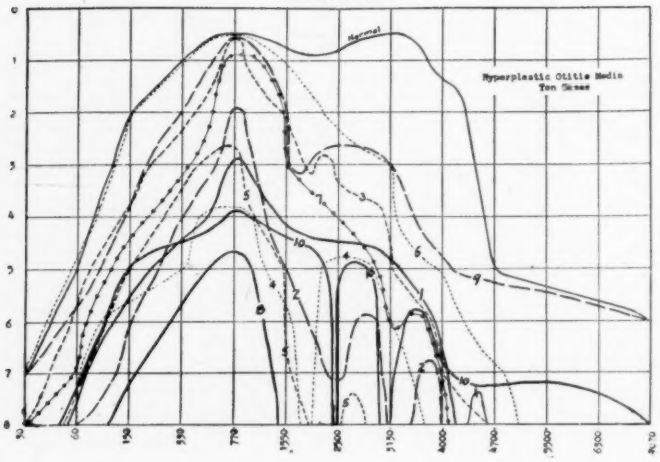

Normal
Hyperplastic Otitis Media
Ten Cases
0
1
2
3
4
5
6
7
8
50
60
150
550
770
1750
2800
3150
4000
4700
5500
6500
20,70

PLATE XXI.

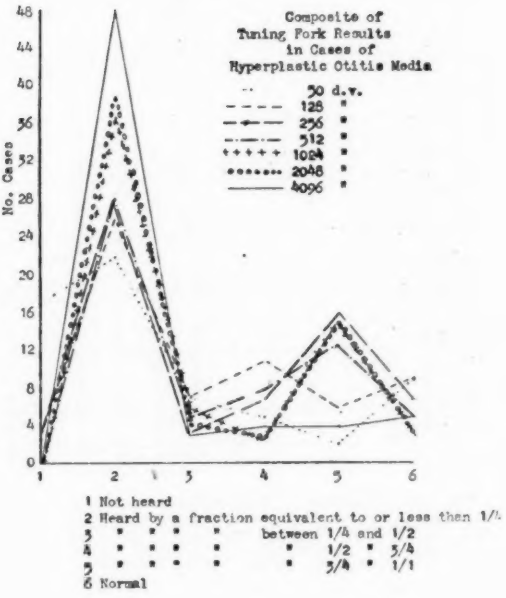
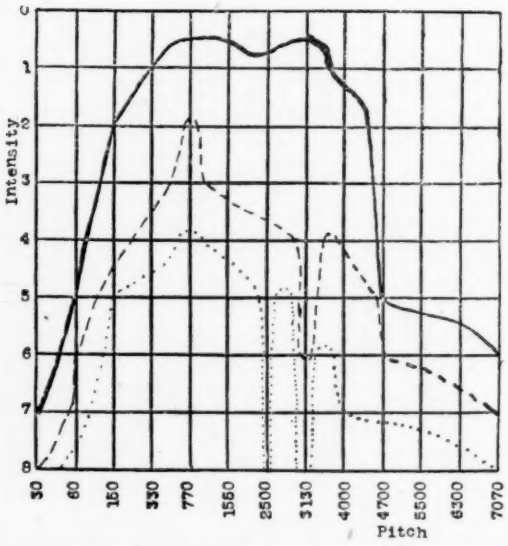

Composite of
Tuning Fork Results
in Cases of
Hyperplastic Otitis Media
50 d.v.
128 "
256 "
512 "
1024 "
2048 "
4096 "
No. Cases
0
4
8
12
16
20
24
28
32
36
40
44
48
1
2
3
4
5
6
1 Not heard
2 Heard by a fraction equivalent to or less than 1/4
3 " " " " between 1/4 and 1/2
4 " " " " " 1/2 " 3/4
5 " " " " " 3/4 " 1/1
6 Normal

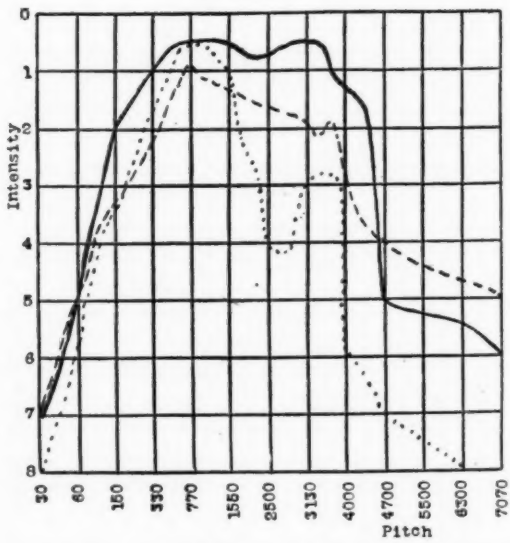
PLATE XXII.



F. K., age 27. Diagnosis: Hyperplastic otitis media. Voice: R. and L., Whisper 2 ft. Rinne: R. and L. negative. B. C. (c^2), R., —2 sec., L. —1 sec. Upper limit, monochord, R., A. C. 17,000, B. C. 17,000; L., A. C. 16,000, B. C. 17,000.

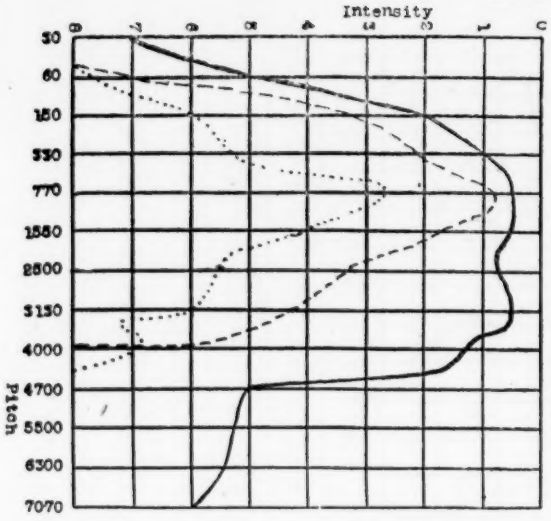
Forks 50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R..... 0	0/8	2/12	4/24	6/36	12/48	15/60
L..... 0	0/8	1/12	3/24	3/36	6/48	8/60

PLATE XXIII.

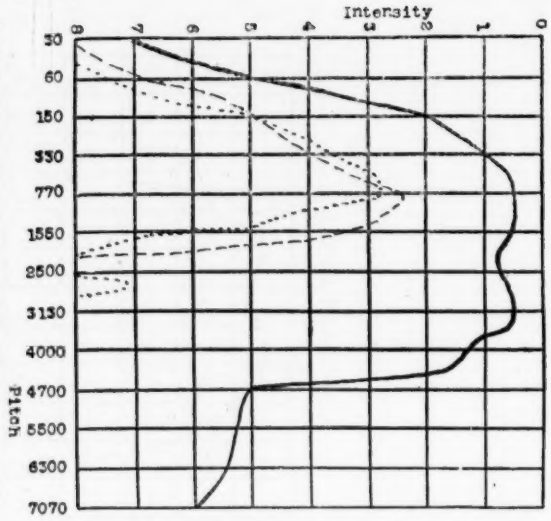


Mrs. C. J. L., age 25. Voice: R., Wh. 5 ft., Sp. 15 ft.; L., Wh. 10 ft., Sp. 30 ft. Diagnosis: Hyperplastic otitis media. B. C. (c^2), R., normal; L., —5 sec. Rinne: R. neg., L. pos. Upper limit, monochord, R., A. C. 17,000, B. C. 19,000; L., A. C. 18,000; B. C., 20,000.

PLATE XXIV.

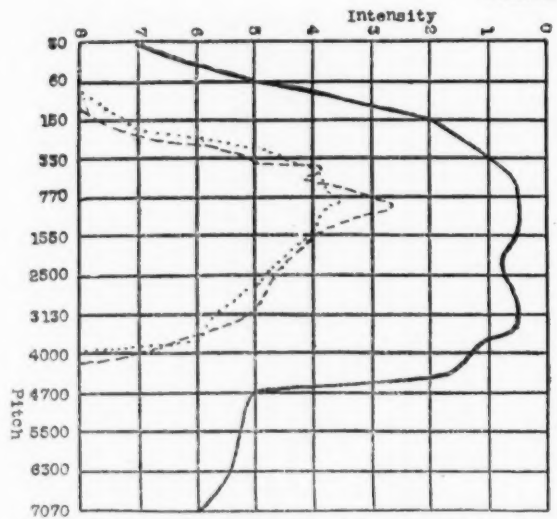


H.S., age 62. Diagnosis: Hyperplastic otitis media. Voice: R., Wh. 1 ft., Sp. 3 ft.; L., Wh. 6 in., Sp. 18 in. Rinne: Positive each ear. B. C.: R., increased 2 sec., L., decreased 5 sec. Upper limit: Monochord, not heard; Galton whistle, not heard; Koenig cylinders, R. and L., 21,845 v. s.



C. P., age 62. Diagnosis: Hyperplastic otitis media. Voice: R., Wh. 15 ft., Sp. 45 ft.; L., Wh. 6 ft., Sp. 20 ft. Rinne: Positive each ear. B. C.: R., —5 sec., L., —12 sec. Upper limit, monochord: R., A. C. 13,000, B. C. 15,000; L., A. C. 14,000, B. C. 16,000.

PLATE XXV.

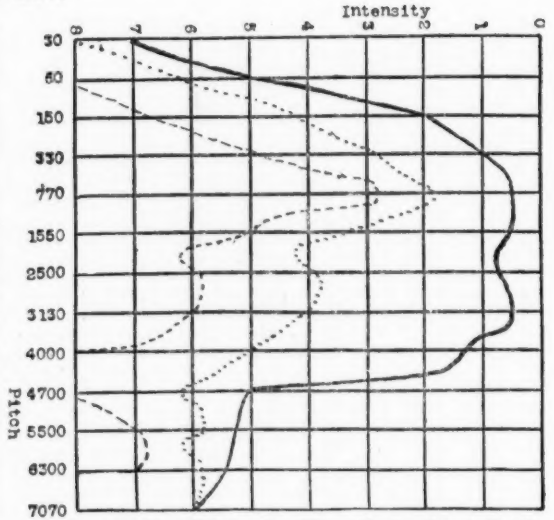


L. D., age 25. Voice: R., Wh. 6 in., Sp. 18 in.; L., Wh. 4 ft., Sp. 10 ft. B. C. (c^2), R. normal, L., increased.

Forks:

50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R...	0	1/8	1/12	2/24	8/26	5/48	2/60
L...	2/4	6/8	8/12	10/24	20/36	24/48	30/60

Upper limit: Monochord, R., A. C. 17,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000.



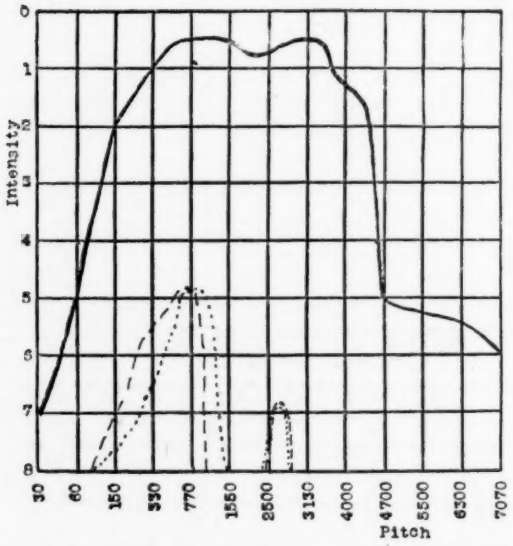
H. B., age 20. Voice: R., Wh. 2½ ft., Sp. 5 ft.; L., Wh. 2½ ft., Sp. 4 ft. B. C. (c^2), R., —4 sec., L. —6 sec.; (c^1), R., —4 sec., L. —5 sec.

Forks:

50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R....	0	1/12	1/18	1/24	1/36	2/48	1/60
L....	0	1/12	1/18	1/24	1/36	2/48	1/60

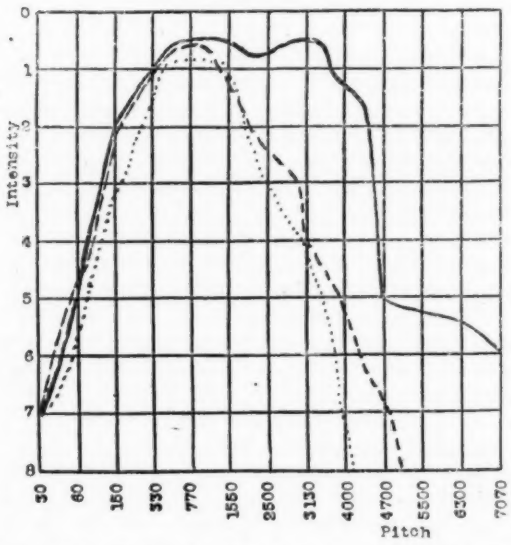
Upper limit: Monochord, R., A. C. 15,000, B. C. 17,000; L., A. C. 13,000, B. C. 15,000.

XXVI.



H. J., age 21. Diagnosis: Hyperplastic otitis media; Wassermann, 4 plus. Voice: R. Wh. 0, Sp. 11 in.; L., Wh. 0, Sp. 11 in. Rinne: c^2 positive on each side; c^1 negative on each side. B. C. (c^2) —14 sec. each side; (c^1) —6 sec. each side. Upper limit: Monochord, R., A. C. 8,000, B. C. 9,000; L., A. C. 8,000; B. C. 9,000.

PLATE XXVII.



J. C., age 48. Voice: R., Wh. 9 ft., Sp. 45 ft.; L., Wh. 8 ft., Sp. 40 ft. Rinne: Positive each side. B. C. (c^2), R., —6 sec., L. —4 sec.

Forks 50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R... 4/4	7/8	10/12	20/24	30/36	40/48	0/60
L... 4/4	8/8	12/12	24/24	30/36	40/48	0/60

Upper limit: Monochord, R., A. C. 11,000, B. C. 12,000; L., A. C. 12,000, B. C. 13,000. Diagnosis: Hyperplastic otitis media.

XL.

OBSERVATIONS ON HAY FEVER.

By I. Chandler Walker, M. D.,

Boston.

The symptom complex to which John Bostock in 1819 gave the name hay fever may attack an individual at various seasons of the year, or may be present more or less continuously throughout the entire year. The former is called seasonal hay fever, and the latter is called perennial hay fever. Seasonal hay fever is a very common complaint, and it has been estimated that one per cent of the population of the United States is so afflicted. Perennial hay fever is much less common. However, if it is proper to include those cases who are subject to frequently repeated head colds and those who have vasomotor rhinitis, both of which conditions are not very unlike perennial hay fever, it then becomes a very frequent complaint.

PERENNIAL HAY FEVER.

Perennial or all the year hay fever is very often caused by the inhalation of the proteins of the hair, dandruff or skin dust of the horse, dog, cat and other fur bearing animals, such as pets and fur wearing apparel. The same holds true for the feathers of the chicken and goose, for example, feather pillows, and feathered pets, such as parrots. The inhalation of plant pollens at a definite season may predispose to a perennial hay fever, and a seasonal hay fever, caused by pollens, such as timothy, grass and ragweed, may become an all the year condition due to other superimposed causes. The inhalation of the flour of the cereal grains is not infrequently a cause of perennial hay fever; such instances are confined to bakers, cooks and those who handle the various types of flour and ground up grain. In fact, the inhalation of any kind of organic dust may cause perennial hay fever; instances of this are the inhalation of face powders and of boxwood and organe wood dust by jewel polishers, etc. Therefore the patient's occupation, habits and surroundings may have a bear-

ing on the cause of the symptoms in this inhalation type of perennial hay fever.

Another group of causes of perennial hay fever we designate as the ingestion type, and this concerns the eating of food proteins. Although in my experience eggs, wheat and milk are the usual food proteins that are found to cause this condition, tests should be made with all of the common foods that the patient is continuously or frequently in the habit of eating. Among the common foods that seem essential to test routinely are egg, wheat, milk, rice, corn, oat, potato, pea, bean, beef, lamb, pork and chicken; additional foods may be determined from the patient. Examples of unusual foods causing perennial hay fever follow: A woman had hay fever from eating raw carrots which she craved and ate every day; another had hay fever whenever she ate celery, and a third patient noticed that among other foods onions caused hay fever.

The above mentioned two types of causes of perennial hay fever, namely, the inhalation and the ingestion types, may be determined by means of skin or cutaneous tests, the technic of which follows. A number of small cuts, each about an eighth of an inch long, are made on the flexor surfaces of the forearm. These cuts are made with a sharp scalpel, but are not deep enough to draw blood, although they do penetrate the skin. On each cut is placed a protein and to it is added a drop of tenth normal sodium hydroxid solution to dissolve the protein and to permit of its rapid absorption. In the case of pollens, animal hair and feathers a concentrated solution of pollen protein or animal hair extract may be used without the addition of sodium hydroxid. At the end of half an hour the proteins are washed off and the reactions are noted, always comparing the inoculated cuts with normal controls on which no pollen was placed. A positive reaction consists of a raised white elevation or uticarial wheal surrounding the cut. The smallest reaction that we consider positive must measure 0.5 cm. in diameter.

The treatment of the above mentioned two types of perennial hay fever, namely, the inhalation and ingestion types, consists chiefly in the omission of the offending protein as determined by the positive skin test. Therefore, treatment

may consist of the omission of a food from the diet or the avoidance of domestic animals and pets, or discarding the feather pillows, or a change in occupation, or discontinuing the use of a certain face powder, etc. Since the avoidance of exposure to animals is not always possible and the avoidance of exposure to common pollens is rarely possible, desensitizing treatment may be given for the former and should be given for the latter. This method of treatment will be described later on, when we discuss seasonal hay fever.

A third type of perennial hay fever we call infectious type because bacteria seem to be the prominent cause. This type is determined by the process of exclusion, and it comprises those patients who not only fail to give a positive cutaneous test but also have no demonstrable rhinologic cause. In other words, cutaneous tests and rhinologic examination have failed to determine the cause of symptoms. For the treatment of this infectious type of perennial hay fever we have to resort to vaccines. Although stock vaccines sometimes give good results, it is best to use autogenous nasal vaccines which are prepared by inoculating the patient's nasal secretion either into dextrose bouillon or into blood agar plates. Vaccine treatment should not be given oftener than at five day intervals and preferably only at weekly intervals, and the dose of vaccine should be gradually increased.

A fourth type of perennial hay fever has already been casually mentioned and concerns the rhinologist. Among the various causes may be mentioned the presence of polyps, adenoids, sinus infection, etc. The rhinologist will determine the cause and treatment of this type; when, however, he fails, autogenous nasal vaccines are worth trying and often benefit.

Dr. Goodale has called attention to a pseudohay fever due to the fragrance of certain heavily scented flowers. It seems to me that this pseudo type of hay fever is due to many causes which may be classified as mechanical, chemical, odorific and thermal. Among the mechanical causes any kind of dust is the most frequent cause, more especially sweeping dust and hay dust; fine powder, such as talcum and the like, is also a frequent cause. Among the chemical irritants, soap powder, lye and ammoniac fumes are very frequent causes. Among the odorific irritants, heavily scented perfumes, face pow-

ders, musty air and stable odors are frequent causes. Thermal irritants concern sudden changes of temperature, as in going from warm air to extreme cold, from moist air to very dry air, and exposure to drafts; a very frequent history is that of a paroxysm of sneezing, with or without running of the nose, on retiring and on arising. The mechanism seems to be a reflex due to the sudden exposure of the warm and protected skin of the body to cold air, as in getting out of bed and in undressing, during which acts the warm body surface is suddenly and momentarily exposed to cool air; in other words, there is a mild chilling of the body surface. The same mechanism holds for many who take cold easily. Some of these patients are sensitive to some type of protein which may have rendered their nasal mucous membranes sensitive to these irritants; others are not sensitive to proteins. Appropriate protein treatment for those who are sensitive usually relieves these symptoms, no matter what the irritant may be, and occasionally autogenous nasal vaccines will benefit or relieve the nonsensitive individual.

Therefore, to summarize, I think that the following procedures offer the best results in the determination of the cause and treatment of perennial hay fever: First do routine cutaneous tests with the common food proteins, common pollen proteins, and domestic animal hair proteins; the need of testing with unusual food proteins, face powders and other unusual types of proteins depends upon the customs and habits of the individual in question. A positive test followed by omission of the protein yields satisfactory results.

With those patients in whom cutaneous tests fail to determine the cause, an examination of the nasal cavities and sinuses may reveal the cause. Autogenous nasal vaccines often have to be given even though previous examinations and tests have or have not revealed a cause. The same procedure of testing, rhinologic examination and vaccine therapy may be applied to the condition of frequent colds. With the vasomotor rhinitis cases I should put rhinologic examination first, then if necessary cutaneous tests and autogenous vaccines. The possibility of a pseudohay fever must be borne in mind, and this may be a primary condition or secondary to other causes.

SEASONAL HAY FEVER.

For convenience we classify or divide seasonal hay fever into three groups, namely, spring, summer and autumn. The spring type concerns those who have symptoms during February, March, April and May, and the causative agent is usually the pollens of trees. The summer type concerns those who have symptoms during late May, June and July, and the causative agent is usually the pollens of grasses. The autumn type concerns those who have symptoms during August and September, and the causative agent is usually the pollen of ragweed; dwarf ragweed in the East and giant ragweed in the West. Naturally the season of pollination of these various plants may vary according to the locations. However, the history of the patient will tell you the time of year when symptoms are present, and it is up to you to determine the prevailing causative pollen. What I shall tell you applies to the New England states, and this will give you a working knowledge for your respective localities. In fact, I am inclined to believe that the causative pollens do not vary very much for different localities, but the time of pollination of the same plant does vary in different localities.

HAY FEVER CAUSED BY THE POLLEN OF TREES.

The first pollen season begins in February and continues into May, during which time various trees pollinate. Silver maple often pollinates in February; the other maples, birches, willows and hazel nut pollinate in late March or early April; the poplars, juniper, cottonwood and elm pollinate during April; the oaks, ash, bayberry and hickory pollinate during May; the pines pollinate in late May and early June, and the fruit trees pollinate in May. I have observed twelve patients who were sensitive to and had hay fever from the pollen of trees. One patient had hay fever caused by the pollen of apple blossoms, and he was free from symptoms following preseasonal treatment with its pollen extract. One patient, who was sensitive to the pollen of the oak and maple, and another, who was sensitive to the pollen of the willow, were both free from symptoms following preseasonal treatment with these pollen extracts. Patients not treated were those sensi-

tive to the following tree pollens: one patient, to poplar tree pollen; one, to pine tree pollen; two, to ash; one, to willow; two, to both willow and poplar, and one to willow, poplar and ash. Since the season of pollination of the individual trees continues only from a few days to two weeks at the most, it does not seem essential that treatment be given. However, treatment may be successfully given for tree pollens in the same manner as for other pollens for which treatment will be detailed later on.

CAUSES OF THE SUMMER TYPE OF HAY FEVER.

Patients who have hay fever during late May, June and July, the so-called rose cold period, are exposed to many kinds of pollens, but actually the pollens of only the grasses cause much trouble. In the New England States some of the compositæ, the most important of which is daisy, pollinate at this time, but for reasons given later on daisy pollen rarely, if ever, is a cause of hay fever. Such plants as the lilies, sorrel, buttercup and others pollinate at this time, but when tested no patient has been found to be sensitive to these, and if they ever cause hay fever it is because of immediate nasal contact with the blossom. The rose which pollinates at this time is rarely a cause of hay fever, since it is not a wind pollinated plant. Since, however, rose is commonly suspected to be the cause of hay fever or rose colds, and since the pollen of rose is occasionally the actual cause, skin tests with rose-pollen frequently have to be done, if for no other reason than to satisfy the patient. So far I have had reason to treat only three rose cases.

The cause of hay fever during June and July in the New England States is practically limited to the pollens of the grass family. Lawn grass begins to pollinate early in May, as a rule, but since it is rare for hay fever to begin early in May, lawn grass pollen is probably rarely, if ever, the chief cause of hay fever. Since, however, lawn grass continues to pollinate throughout the summer, even when it is repeatedly mowed closely, with those patients who are sensitive to the pollens of other grasses and are either not treated or are insufficiently treated with the particular grass pollen that is the chief cause of hay fever, exposure to lawn grass

pollen may be a complicating cause or may aggravate the symptoms of hay fever. Orchard grass pollinates during July, but this type of grass grows in more or less secluded places, and it is not commonly encountered. I have not tested a patient who reacted strongly to orchard grass. Since corn is a member of the grass family, and since the table variety pollinates during July, it must be considered among the possible causes of early hay fever. Corn pollen is very heavy. It rapidly descends to the ground and is carried by air currents or wind only a few feet. Therefore, intimate exposure would be required to produce symptoms. Furthermore, only rarely would a person be unable to avoid it. Therefore, corn pollen must rarely, if ever, be considered a cause of hay fever in the New England States, where no large acreage exists in any locality as is the case in the West. This same statement is, likewise, true of wheat, oats, barley and rye.

The grasses, then, with which we are concerned in New England are June grass, timothy and redtop, the pollens of which are light and are carried by wind considerable distances. June grass begins to pollinate some years as early as the middle of May; when the season is very late it does not pollinate before the last day or two in May; pollination continues for about three weeks. Timothy and redtop begin to pollinate between the middle of June and the first of July, depending on the season, and pollination continues until the middle or last of July; usually, the season of pollination lasts about six weeks. Therefore, in order to complete the schedule of treatment, mentioned later on in this paper as being desirable, preseasonal treatment with June grass must begin the first of March at the latest, and with timothy and redtop it must begin the middle of March.

CAUSES OF AUTUMNAL OR LATE HAY FEVER.

In the New England States ragweed (ambrosia artemisifolia, or dwarf ragweed) is not the only plant that pollinates during August and September, nor is it the only pollen to which individuals are exposed or with which patients should be tested. Most of the compositæ, such as golden rod, sunflower, golden glow and aster, pollinate during August and September; daisy pollinates during June and July, and dan-

delion in the early spring; giant ragweed is rarely encountered in the New England States. In my experience, however, pollens other than ragweed rarely, if ever, are the chief cause of symptoms during the late hay fever season. I have tested one hundred and seventy patients with the pollens of daisy, golden rod, sunflower and golden glow. Fifty per cent of those tested with these pollens gave a positive reaction with the whole pollen, but none of these gave a reaction with a 1:100 dilution of the pollen; the other 50 per cent failed to react at all with the whole pollen. Therefore, it may safely be stated that the pollens of golden glow sunflower, daisy and golden rod are not primarily causes of hay fever in New England. It should be borne in mind that daisy pollinates during June and July and has finished pollination before ragweed begins, therefore daisy cannot complicate the causes of August and September hay fever. In comparison with ragweed, in only 3.25 per cent of my cases could golden rod pollen be assumed as a possible cause of hay fever. From the investigations of Scheppegrell and others we know that the pollens of golden rod, sunflower, golden glow, daisy and aster are not carried by the wind, and that their pollens are heavy and shed very sparingly, so that for these reasons patients are not exposed to these pollens unless the flowers are kept in the house or unless sensitive patients smell of the flowers; naturally, the patient can refrain from such intimate contact and need not be treated for these.

I have outlined the pollens that may be the possible cause of hay fever at various seasons and I have also described the cutaneous test which when used with these pollens will determine the probable cause. Before treatment may be given it is essential to do cutaneous tests with various dilutions of the pollens or pollen protein in order to determine to which pollen of several the patient is most sensitive and with which pollen the patient should be treated, and with what dilution of pollen that gives a reaction on the skin, but treatment should be begun with the strongest solution that fails to give any reaction whatever.

These solutions I make as follows: To 0.5 gm. of the dry pollen is added 44 cc. of sterile physiologic sodium chlorid solution; the mixture is shaken thoroughly at frequent intervals

for twenty-four hours, after which enough absolute alcohol (6 cc.) is added to the mixture to make the alcoholic content 12 per cent. Again, the mixture is thoroughly shaken at frequent intervals for twenty-four hours, after which it is centrifugalized at high speed and the supernatant fluid is pipetted off and saved. This supernatant fluid, therefore, consists of the pollen protein dissolved in a 12 per cent alcoholic physiologic sodium chlorid solution and it represents, by weight, 1 part pollen to 100 parts solvent. This 1:100 solution is used as stock, and from it other dilutions, 1:500, 1:1,000, 1:5,000 and 1:10,000 are made, using a 12 per cent alcoholic physiologic sodium chlorid solution as a dilutent. These solutions are used not only for the skin tests but for treatment, and with the addition of a small crystal of thymol they keep for many months in a cool place.

METHOD OF TREATING PRESEASONALLY WITH POLLEN EXTRACTS.

The first treatment consists of from 0.1 to 0.2 cc. of that dilution next higher than the one which gave a positive skin test, or, in other words, the first dose is 0.1 cc. or 0.2 cc. of the strongest dilution which failed to give any skin reaction whatever, no matter how slight. With my pollen extracts the majority of patients whom we treated gave a more or less positive reaction with the 1:10,000 dilution, therefore, the first treatment consisted of 0.1 cc. or 0.2 cc. of the 1:20,000 dilution. Treatments are given subcutaneously once a week, and each week the amount of the extract is gradually increased, so that as the treatment progressed, stronger and stronger dilutions were used, until one or more doses of the 1:100 dilution were given. As an example, I will give what I have found by experimentation to be the best outline for a patient who gives a more or less positive skin test with a 1:5,000 dilution of pollen extract; 1:10,000 give 0.15 cc.; 1:5,000 give 0.15 cc., 0.25 cc., 0.35 cc., 0.45 cc., 1:1,000 give 0.15 cc., 0.25 cc.; 1:500 give 0.15 cc., 0.25 cc., 0.35 cc., 0.45 cc.; 1:100 give 0.15 cc., 0.2 cc., 0.25 cc. Each dose was given preferably at weekly intervals and never oftener than once every five days.

This schedule of treatment calls for fourteen inoculations. However, for some reason or other, modifications frequently

have to be used. Often a patient is so sensitive to the pollen that a 1:10,000 dilution gives a slight reaction, thus necessitating an initial dose of 0.15 cc. of 1:20,000 followed by possibly two doses of 1:10,000. Often it happens that a patient has considerable local or general reaction following some one treatment in the schedule, thus necessitating the repetition of that particular dose before the next increase may be given. More often the patient presents himself for treatment too late to complete the scheduled series of treatments before the onset of pollination so that for preseasonal treatment alone, some of the final treatments in the schedule must be omitted. This schedule is often modified purposely with certain individual cases. For instance, in some cases the second treatment with the 1:1,000 dilution, namely, 0.25 cc., is omitted, and in some cases instead of giving 0.15 cc. of the 1:100 dilution, when this happens to be the final treatment that the patient is to receive because of onset of pollination, a fifth treatment with the 1:500 dilution, namely, 0.55 cc., is often substituted, and even a sixth treatment with the 1:500 dilution, namely, 0.65 cc., is sometimes given. These larger doses of 1:500 approximate the amount of protein in 0.15 cc. and 0.2 cc. of the 1:100 dilution, therefore, the fifth and sixth treatment with the 1:500 dilution, as outlined, is practically the equivalent of giving 0.15 cc. and 0.2 cc. of the 1:100 dilution. Since by far the great majority of patients are treated from three to five times with the 1:500 dilution, and since this number of treatments has given fairly satisfactory results, I consider this number of treatments, which consists usually of a total of ten, as worth giving, although a continuance of the schedule beyond three doses of the 1:500 dilution is most desirable, and giving less than three treatments with the 1:500 dilution is undesirable.

Since in the New England States the majority of autumnal hay fever patients have their first symptoms between August 10 and 20, during which time the compositae, chiefly ragweed, begin to pollinate, in order to complete the above schedule just previous to the onset of symptoms and pollination patients must begin treatment between the last week in April and the first two weeks in May. Beginning treatment the first week in June permits of giving from three to five treatments with the 1:500 dilution. Likewise, since the early type of hay

fever, or socalled rose cold, which is usually caused by the grasses, begins the last few days of May or the first of June, treatment for this type of hay fever should begin about the first of March, and the starting of treatment as late as the first of April will not permit of more than three to four treatments with the 1:500 dilution, according to the schedule outlined. Naturally, in localities outside of New England, these seasons would differ, and the beginning of treatment consequently would vary, also the causative pollen must differ.

My conclusions, based on a four years' experience with the treatment of June and July hay fever, are: While the pollen of timothy grass is the chief cause of hay fever, and sufficient treatment with it alone gives excellent results, it is necessary to test all patients with the pollens of redtop, June grass and rose, since an occasional patient is more sensitive to one of these pollens than to timothy pollen, and, therefore, such a patient may need treatment with one of them rather than with timothy pollen. When the same patient, as is frequently the case, is equally sensitive to the dilutions of the pollen extracts of timothy, redtop and June grass, it is advisable to treat the patient with timothy pollen extract alone because there is sufficient evidence that treatment with timothy pollen will protect against redtop pollen exposure, provided one or more treatments are given with the 1:100 dilution of the pollen extract; timothy and redtop pollinate at the same time, and redtop is less prevalent than timothy. Sufficient treatment with timothy pollen extract seems to protect also against June grass pollen exposure, but since, as is often the case, June grass pollination is well advanced or even sometimes completed before timothy pollination begins, the patients being treated with timothy have not had sufficient treatment with timothy pollen to protect them completely against June grass pollen, consequently some of these patients will have more or less symptoms from June grass exposure. Treatment with both timothy pollen and June grass pollen extracts together will not only be insufficient to protect against early June grass pollination, but the combination of the two pollen extracts necessarily retards and diminishes the amount of treatment that might be given with timothy alone. Therefore, I am opposed to treatment with pollen mixtures, except on rare

occasions, and in the case of June grass it is better to take a chance on a late season with timothy protection or even an early season with few symptoms than to treat with it in conjunction with timothy when timothy treatment is essential. Rarely one meets with a patient who has hay fever only from early in May to the middle of June; in such a case treatment with June grass alone is advisable and usually sufficient. Rarely it is necessary to treat with rose pollen or some other grass than those mentioned. However, the cutaneous tests will determine this for you.

I have treated, according to the scheme already outlined, 115 grass cases of hay fever and the results from the first year's treatment follow: Twenty-eight and seven-tenths per cent were free from symptoms, 21.7 per cent were practically free, 23.5 per cent were 75 per cent benefitted, 21.7 per cent were 50 per cent benefitted and 4.3 per cent were not benefitted. Therefore, from the first year's treatment given preseasonally for grass hay fever, you may expect equally good results. Treatment of the same patient in successive years yields even better results, therefore, you can give your patients a better prognosis the second year than the first, provided as much or more treatment is given the second year as was given the first year.

My conclusions, based on four years' experience with the treatment of August and September hay fever, are that ragweed is practically the only pollen with which treatment is necessary; dwarf ragweed in the East and either dwarf or giant as the case may test in the West. In various localities there may be some pollen other than ragweed that is a causative factor. I have treated 420 cases with ragweed pollen with the following results for the first year's treatment: Twenty-one and five-tenths per cent were free from symptoms, 17.5 per cent were practically free, 25.5 per cent were 75 per cent benefitted, 29 per cent were 50 per cent benefitted, 6.5 per cent were not benefitted. Therefore, from the first year's treatment you may expect equally as good results and from successive years' treatment you may expect even better results.

The permanency of benefit from treatment seems to depend largely upon the individual and to some extent upon a large amount of treatment which renders the patient non-sensitive.

After two or three years of consecutive treatment the majority of patients will continue free or practically free from symptoms for another two or three years without treatment. After this period of time symptoms return more or less gradually. Occasionally a patient will be free only one year before symptoms return and occasionally symptoms will be as severe the first year that treatment is omitted. Rarely one season's treatment will protect for several years and I think treatment every other year with some individuals will keep the patient quite free from symptoms. As a rule, I feel it best to give two or three successive years' treatment before permitting omission of treatment.

DURING THE SEASON OR CURATIVE TREATMENT WITH POLLEN.

Frequently patients present themselves for treatment during their hay fever attack, and although pollen treatment at this time does not seem to be very logical, on the basis of anaphylaxis, the patient often will insist on taking the chance. Pollen treatment during the season does not seem logical because the patient is being injected with the pollen which is causing symptoms at the same time that he is being exposed to the pollen present in the air which he is inhaling. The danger resulting from large doses of the injected pollen is obvious because of an overdose, due to the combination of the injected pollen and the inhaled pollen. Therefore, in order that during the season treatment should be beneficial, the patient must be injected with minute amounts of the pollen extract in order to diminish artificially a few of the patient's antibodies, thus leaving a smaller number of antibodies in the patient for combination with the pollen antigen that is inhaled. If too much pollen extract (antigen) is injected, the patient should have symptoms due to over treatment alone, or he should be made worse, due to the injection of pollen extract (antigen) superimposed on the inhalation of pollen (antigen). It is evident that, on the basis of anaphylaxis, during the season treatment is hazardous, and although the skin test is the best guide as to the proper treatment, there is no way of controlling the amount of pollen that the patient may inhale.

The results from during the season treatment with grass pollen were: Of twenty-five patients treated, none were free

from symptoms; seven, or 32 per cent, were practically free; four, or 18 per cent, were 75 per cent benefitted; three, or 13 per cent, were 50 per cent benefitted; the same number were 25 per cent benefitted, and five, or 22 per cent, were not benefitted. Since no patients were entirely relieved of symptoms, and since a large percentage were not benefitted, it is evident that preseasonal treatment insures better results by far than does during the season treatment. If preseasonal treatment is absolutely barred, during the season treatment is worth trying, provided it is given with sufficient care and the skin test is used as a guide to the initial treatment.

The results from during the season treatment with ragweed were: Of twenty-seven patients, three, or 11 per cent, were free from symptoms; one patient, or 3 per cent, was practically free; three patients, or 11 per cent, were 75 per cent benefitted; seven, or 25 per cent, were 50 per cent benefitted; one patient, or 3 per cent, was 25 per cent benefitted, and twelve patients, or 44 per cent, were not benefitted. Therefore, although a few patients were free from symptoms, and as many more were 75 per cent benefitted, the number who were only 50 per cent benefitted was as great as the number who were more than 50 per cent benefitted, and nearly half of the whole series were not benefitted at all. On comparing these results from during the season treatment with the results from preseasonal treatment, it is evident that for late hay fever by far the best results are obtained from preseasonal treatment, and it is questionable whether during the season treatment is even worth giving when it is taken into consideration that there are localities to which patients may go where ragweed does not exist.

PRECEDING AND DURING THE SEASON TREATMENT WITH POLLEN.

Some patients present themselves a few weeks previous to their season of symptoms, that is, they apply for treatment too late for preseasonal treatment alone and too early for during the season treatment. Rather than let them wait until their symptoms begin and then give them during the season treatment it is best to immediately begin treatment and con-

tinue the treatment on through their period of symptoms. This method of treatment yields better results than does the during the season treatment, but not as good results as the preseasonal treatment.

DURING THE SEASON TREATMENT WITH BACTERIA.

Occasionally when preseasonal pollen treatment fails, treatment during the season with autogenous nasal vaccine or a mixed streptococcus vaccine will benefit. The reason for such treatment is that it is quite possible that ragweed pollen exposure may in some cases cause such severe irritation of the mucous membranes that ever present bacteria may, either alone or together with ragweed pollen, be a cause of hay fever symptoms.

OTHER POLLENS THAT MAY CAUSE HAY FEVER.

It has been shown in this paper that the chief causes of hay fever occurring in the New England States were the pollens of ragweed, timothy and June grass; that an occasional cause is the pollen of the rose and redtop grass and of various trees. There is, however, almost an unlimited list of pollens that may at any time, but probably rarely, cause hay fever. Therefore, for those who treat a large number of hay fever patients and for those who fail to obtain satisfactory results from treatment with the common pollens mentioned, it is advisable to have a very extensive assortment of pollens. As a rule, however, it would seem to be sufficient to warn the ragweed patients that they should not smell the golden rod, golden glow, sunflower, poppy, aster, chrysanthemum and the like that pollinate during the ragweed season. The grass cases may be warned to avoid close contact with clover, lilies, daisy, dandelion, rose, lawn grass, orchard grass, corn and the like that pollinate during the grass season. In certain localities in this country it may be necessary to treat with the pollen extracts of the various grains, with sunflower, sage brush, or what not, but this necessity is not universal and is limited to the various localities where exposure to these particular pollens is unavoidable.

OTHER PARTS OF PLANTS THAT MAY CAUSE HAY FEVER.

The leaves of certain plants and trees may cause hay fever. The under surface of some leaves has a fine hairy growth, and these fine hairs may cause hay fever. I have observed one patient who had hay fever from and was sensitive to the hairs of the willow tree leaf; treatment with an extract of these leaves prevented hay fever. Another patient was sensitized to and had hay fever from the plantain lead. It is quite probable that the plane tree, the common shade tree in London, may cause hay fever. Such instances, however, are extremely rare and need not be considered, as a rule.

FOODS MAY CAUSE SEASONAL HAY FEVER.

Seasonal hay fever, due primarily to foods, has not come to my notice. However, during their season many hay fever patients find that certain foods aggravate their symptoms, whereas these same foods may be eaten without symptoms at a time when they are not accustomed to have hay fever, and they can eat the foods during their hay fever season, provided they have had sufficient pollen treatment to protect them against the particular pollen that primarily causes their hay fever. These foods are usually the fruits, and they have no apparent relationship to the causative pollen. Among the usual fruits should be mentioned peach, melon and apple. Green corn and the use of beer and wine often aggravate ragweed hay fever, but they do not aggravate grass pollen similarly. With the peach it is often the skin that causes trouble, whereas the pulp does not. The cooking of the fruits often renders them inert

ANIMAL EMANATIONS MAY CAUSE SEASONAL HAY FEVER.

The patient who is sensitive to animal emanations usually has symptoms on exposure to the animal at any and all times of the year; occasionally, however, such a patient has symptoms only from exposure to the animal during the spring and summer, when the animal is shedding, moulting or perspiring profusely.

BACTERIA MAY CAUSE SEASONAL HAY FEVER.

The possibility of bacterial infection as a complication of or secondary to pollen sensitization has already been discussed,

and the possibility of bacterial infection as a primary cause of hay fever in patients who are not sensitive to pollens must be considered. Each year I examine three or four patients who have seasonal hay fever, but who are not sensitive to any of the pollens; therefore, these patients are treated with their autogenous nasal secretion vaccine.

PSEUDO HAY FEVER.

I have already, under perennial hay fever, discussed the possibility of pseudo hay fever which may complicate a seasonal hay fever as well.

CONCLUSIONS.

Satisfactory preseasonal pollen treatment yields excellent results in seasonal hay fever. By satisfactory treatment is meant five or six treatments with a 1:500 dilution of the pollen extract, or, better still, two or three treatments with a 1:100 dilution of the pollen extract.

When preseasonal pollen treatment fails, sometimes benefit results from during the season treatment with pollens and sometimes from during the season treatment with an autogenous nasal secretion vaccine. During the season treatment with pollens without preseasonal treatment is not very satisfactory, although such treatment for the early type of hay fever is worth doing provided, for some reason or other, it is not possible to give preseasonal treatment.

Treatment that of necessity must begin late preseasonally may be continued on through the pollen season with better results than those obtained by during the season treatment alone, but with much less beneficial results than those obtained by beginning preseasonal treatment early enough to permit of its discontinuance before the season of pollination begins.

Although in the New England States the pollen of ragweed (dwarf variety) is practically always the cause of hay fever (August and September), and the pollen of timothy grass is the cause of probably 90 per cent of the early hay fever (June and July), in other localities other pollens may play a great part in the cause of either type of hay fever. For this reason it is essential that other observers publish their tests, results and treatment in detail in order to learn the causative pollens and proper treatment, in various localities.

In the New England States the pollen of rose and redtop grass occasionally causes early hay fever, and treatment occasionally has to be given with these. The pollen of June grass is a more or less common cause of early hay fever, but when the season of its pollination is very early, unless the treatment is begun very early, too little treatment can be given with it to be of benefit, and when its pollination is very late sufficient treatment with timothy pollen has been given to protect against June grass pollen exposure.

Treatment with a combination of timothy and June grass pollens was not successful in my hands because of insufficient treatment with both pollens; the addition of June grass pollen retarded the amount of treatment that otherwise would have been given with timothy alone. Another illustration of the undesirability of mixed pollen treatment is noted with those patients who have both the early and the late types of hay fever. In these cases, during the month of May and part of June, large amounts of timothy pollen extract should be given together with small amounts of ragweed pollen extract, and the result in these cases is rather poor because there is a tendency to restrict the timothy treatment for fear of producing anaphylaxis from the combination of two pollens to which the patient is sensitive. In other words, treatment with a combination of pollens either diminishes the amount of treatment that is required with one or all of them, or pushing the treatment with the combinations leads to the danger of anaphylaxis For either or both of these reasons, I see no benefit to be derived from mixed pollen therapy. As regards the early type of hay fever, I consider it best to treat preseasonally with timothy or the chief causative pollen and, if necessary, to treat during the season with June grass pollen.

Since intimate exposure to other pollens which may be attributing causes of hay fever, the eating of fruits during the hay fever season, the possibility that bacteria play a part in the cause of hay fever and the exposure to olfactory irritants may all aggravate the symptoms of hay fever, it is remarkable that hay fever treatment is as near perfect as I have tried to present to you.

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XLI.

THE UNPLEASANT END-RESULTS IN WELL PERFORMED TONSILLECTOMIES.

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The tendency of nature to replace tissues removed through accident or design; to restore function through complementary or compensatory action, are well recognized facts to all surgeons. This action is observable in connection with lymphatic tissue, especially when removed in early life. Especially is this to be noted in connection with the faucial and pharyngeal lymphatic tissues. Almost every one of my hearers have seen these facts illustrated in some of their operative cases or in the operative work of some of their equally skillful colleagues. It has seldom been the lot of any operator, however skillful he may be in his operative dexterity, to find all of his cases, one, two or three years thereafter, to have absolutely clear tonsillar fossæ, free entirely of evidences of lymphatic tissue. Even though the operative field may be clear, there is occasionally a case wherein there is an overgrowth of lymphatic tissue in close contiguity to the operative field. I do not wish to be placed in a position of stating that such occurrences are common, after thorough removal of the faucial tonsils, for such is not the case. I wish also to discriminate between those cases in which the tonsils have been thoroughly removed and those which have been imperfectly removed, which latter can be readily differentiated from the types to which I refer. It is a good practice to take advantage, which I always do, and examine every case that I have previously operated upon when the opportunity presents itself.

These few introductory remarks will bring us directly to the consideration of the unpleasant local end-results in well performed tonsillectomies.

The first group will include those efforts of nature to replace lymphatic tissue removed from the tonsillar fossæ by hypertrophy of glandular tissue in the immediate neighborhood.

A. Hypertrophy of glandular tissue in the lateral pharyngeal wall, posterior to the palatopharyngeal fold.

B. Hypertrophy of the lateral borders of the lingual tonsils.

C. Development or hypertrophy of glandular tissue in the palatopharyngeal fold.

In the first of the three groups above mentioned, I do not refer to simple showing of moderate hypertrophy of the follicle usually found in this area, but refer to those distinct tumor-like growths of lymphatic tissue which in their size and conformity resemble miniature tonsils.

In 1910 I saw the first evidence of this type of nature's attempt to replace a removed tissue in close proximity to the destroyed gland. This occurrence took place in a child of ten, upon whom I had several years before done apparently a perfect tonsillectomy. The case had been seen by a colleague, who promptly told the parents that I had not removed the left tonsil. They immediately called the attention of the fact to Dr. S. S. Adams, who referred the child back to me for reexamination. On the reexamination of the child I was surprised to note, in the left lateral wall immediately behind the posterior pillar, a large mass of lymphatic tissue perfectly resembling a faucial tonsil.

In June, 1914, I had a young girl of fourteen come to me on account of an acute inflammation of the left ear. On examining her fauces I found a perfect pair of tonsillar fossæ, but immediately behind the posterior pillar a similar growth of lymphatic tissue, though not quite as prominent as in the case just narrated.

On March 1, 1922, I had a young boy of twelve years referred to me by a distinguished member of this organization. In examining his fauces I found just behind the posterior pillar, in the lateral wall, a smaller but similar mass of hypertrophied lymphatic tissue.

The second group forms a most interesting series. I have seen six cases of this type, two in children and four in adults. The only one occurring following my own operative work was under my observation for aural treatment, twelve years after the removal of his tonsils. The patient stated that as it caused him no annoyance, he preferred not having it removed. In

depressing the tongue, two dark reddish raspberry like masses were observed filling in the lower part of the tonsillar fossæ, giving the casual observer the impression that the lower lobe of the tonsil had not been removed. Depression of the tongue downward and forward caused the masses to move with the tongue, showing the fossæ free and demonstrating their true nature. The other three adults had discomfort and the masses were removed.

In the cases of the two children the condition was much more serious. The first case was that of a French child, male, twelve years of age, who was brought to me to have his tonsils reoperated upon. From his parents I learned that he had had what was thought to be a perfectly successful tonsillectomy done in Paris when he was eight years old. During the past year all of his old tonsillar symptoms had recurred. For several months he had had suffocating attacks at night, which were gradually increasing in their severity. Each night he would be aroused several times through attacks of "night terror." The child was anemic, under weight, and exceedingly nervous.

Examination of the fauces revealed large masses filling in the lower tonsillar fossæ. Depressing the tongue downward and forward caused these masses to follow the tongue and leave their position in the tonsillar fossæ. These masses were irregular, ragged nodular formations of a deep red color. Further investigation demonstrated that the tonsillar fossæ were free of tonsillar tissue and that the presenting masses were outgrowths from the lateral borders of the lingual tonsils. This adventitious tissue was removed. The masses were one and a half centimeter in diameter.

The second case was that of a female child, seven years of age, whose tonsils had been removed when she was four years of age. She had been examined by several physicians in Pittsburgh, all of whom believed that the tonsils had returned. The symptoms were similar but not as severe as in the case above narrated. The view obtained of the fauces and relation of the masses to the lingual tonsil were the same, excepting that the masses were not so large. They were removed.

Deposition of Lymphatic Tissue in Posterior Pillar.—I have seen three cases of large fusiform posterior pillars following

perfectly performed tonsillectomies that had all the normal appearances of tonsillar tissue. Two of the cases I saw on account of acute follicular infection that had the characteristic appearance and all the symptoms that constitute acute follicular tonsillitis. In fact in one, my own case, during the attack I had to carefully examine the fauces before I could convince myself that the tonsillar fossa was clear.

In the healing process of enucleated tonsils, especially when the fossæ are very deep, there is a tendency to fill in the deep cavity by the development of granulation tissues. In most cases this granulating tissue is gradually absorbed in the nature process of repair, and we have a smooth, clean fossa. At other times this tissue undergoes organization, with the appearance here and there throughout its structure of lymphatic nodes. All of us have seen these cases, either in our own patients, or in the patients of our colleagues. There can be no doubt that granulation or true tonsillar tissue can develop in the tonsillar fossæ some months, even years, after a thoroughly well performed tonsillar operation. I have had under my observation on several occasions a woman for a recurrent subacute middle ear inflammation. She had had her tonsils removed in Chicago some five years before I first saw her, and I remember when I first viewed her fauces how I congratulated her upon the perfect result she had secured. I saw her several times at more than yearly intervals and always inspected those perfect tonsillar fossæ. Last spring she reported to me again for a few treatments, and at this time told me she had been having for several months some soreness in the left side of her throat. Imagine my surprise at noting in the lower portion of the left tonsillar fossæ a red inflamed mass nearly as large as the last phalanx ot my little finger.

Allied to the above mentioned group is the redevelopment of islands of tonsillar tissue to large masses of lymphatic tissue in the tonsillar fossæ. These small areas develop some period after the operation. It matters not how well the operation is done. All operators see these sequelæ. I see frequent evidence of this condition in very many cases of the best operators in the country. I have now a boy and a girl in their "teens," under my observation, who present this condi-

tion quite marked who were operated upon by a surgeon whom you and I recognize as the best operator in the United States. I had several years ago a case of this type that quite surprised me in several ways. The recurrence was no larger than a small pea, but displayed two follicles containing cheesy material. I had seen this child two years after a most perfect tonsillectomy, and all was perfect in the faucial fossæ. I never did a more perfect operation. Four years after the original operation I was confronted with the patient and the rather indignant mother, who stated that the family physician had said that I had never removed the left tonsil. The child lost weight, her color was bad, no appetite, and she had grown listless, tiring quickly. I examined the fauces and saw the small, pealike excrescence of lymphatic tissue in the bottom of the fossæ. While carrying on a running conversation I cocainized the excrescence and quickly pinched it off before they realized my intention. Within a few weeks the child returned to the normal. I mention this case to demonstrate that we must not always consider these cases too lightly.

Adhesion or shortening of the anterior pillar by its adherence to the dorsum of the tongue, and the adhesion of the lower lateral wall of the fossæ to the side of the tongue form another group. This group of accidents gives rise to very uncomfortable symptoms, if we are to accept the statements of patients. Extreme care is the watchword to prevent this accident. Occasionally it will occur in spite of our best endeavors.

Injuries of branches or the trunk of the hypoglossal nerve give rise to annoying symptoms. I have had one such case in my own practice and others in the practice of my colleagues.

My object in presenting this review of the unpleasant local end-results of well performed tonsillectomies is to make us more careful in our work along these lines so as to avoid as many of these pitfalls as possible. To make all endeavor to see our patients who have been operated upon six months or a year after the operation. To be more charitable to those with whom we work in a common field. Do not accuse any other man of having made an incomplete operation—no one can make such a statement with honesty unless he saw the operation performed.

XLII.

A CONTRIBUTION TO THE STUDY OF BRAIN ABSCESS.

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In reviewing the subject of brain abscess one is struck by the scarcity of symptoms observed, and the following paper represents an attempt to ascertain their relative frequency and diagnostic value. Most of the case histories I have been able to study are somewhat meager, and in many cases one cannot assume that the lack of specific mention of certain symptoms means that they were absent. They were probably not observed. Yet, even in the case where negative findings are definitely stated there is usually relatively little upon which to base a definite diagnosis. The clinical picture of brain abscess presents a wide variety of symptoms and signs, but this picture is a composite one, and one can never expect to find more than a few symptoms in any one case and they are often fleeting or nebulous. Symptoms of increased intracranial pressure are, as a rule, fairly definite. These, however, may be due to extradural abscess, cerebral abscess, cerebellar abscess, tumor or meningitis. The more acute the condition the more violent are the symptoms. Thus, in tumor, which generally takes months to grow, although the mass may be large, signs of increased pressure may not be so marked as they are in the case of abscess, or still more in the case of meningities, where the evolution may occur within a few days.

In this paper I propose to give an analysis of the histories of 67 cases of otogenic brain abscess, obtained from the literature. Detailed notes of a recent case of cerebellar encephalitis and one of temporosphenoidal abscess are also added.

FREQUENCY OF OCCURRENCE.

Taking the figures supplied by the Royal Infirmary of Edinburgh reports for the five years 1914-18 inclusive[1] we find that out of a total of 3,918 cases of middle ear suppuration,

including dried up cases but not including acute or chronic catarrh, there were 85 (2.19 per cent) cases with intracranial complications. Of these 27 (0.58 per cent) occurred in acute cases and 58 (1.4 per cent) in chronic cases. Out of these 85 cases 15 were brain abscess. The frequency then of the occurrence of brain abscess in relation to otitis media is 15 to 3,918, or 0.38 per cent, that is, about 4 per thousand. It will be noted incidentally that intracranial complications occur more than twice as often in chronic otitis media as in acute. Logan Turner in 1909[2] mentions 14 intracranial complications in 532 cases of middle ear suppuration (2.6 per cent). Out of these three were brain abscesses (0.5 per cent). Mygind[3] in 207 cases of otogenic intracranial disease found meningitis 141 times, sinus thrombosis 106 times, brain abscess 42 times and subdural abscess 19 times. Multiple complications were twice as common in chronic cases as in acute (48 to 25).

TRACKS OF INFECTION FROM MIDDLE EAR.

In the case of temporosphenoidal abscess, probably by far the greater number are caused by direct spread of infection upwards through the tegmen tympani. In most of the recorded cases which were available for study the tegmen was found to be eroded and the dura covered with granulations. Here a localized patch of meningitis is set up, the pia arachnoid becomes adherent to the dura and the process spreads into the brain itself, forming a subdural abscess. Vessels on the surface of the brain become thrombosed and cause septic softening in the white matter. In other cases, however, the tegmen is not eroded, and when the dura is exposed it appears healthy except for increase of tension. The brain is not by any means always adherent to the membranes when there is an abscess underneath. The surface frequently looks quite normal. In such cases it is more than likely that the abscess originates in a small septic embolus. That part of the temporosphenoidal lobe which lies over the tegmen represents one of the boundary zones in the cerebral circulation, where the regions supplied by the middle and inferior cerebral arteries meet. (Fig. I.) The vessels here are hence all of small caliber and easily permeable. The veins of this region require special study.

The great bulk of the venous blood from the brain leaves

the skull by the sigmoid sinus and internal jugular vein of each side. However, there are numerous accessory outlets which act as safety overflows. These are the mastoid foramen with its emissary vein and the posterior condylar foramen, both from the lateral or sigmoid sinus. The inferior petrosal sinus leaves the skull through a separate compartment of the jugular fossa.

There is also a free communication between the cavernous sinus and the pterygoid plexus on each side through the foramen ovale. The superior petrosal sinus, which is relatively small, forms a communication between the cavernous sinus and the genu of the lateral sinus on each side. The blood flows away from the cavernous sinus towards the sigmoid. However, owing to its oblique entrance into the latter, its flow must necessarily be retarded, and under varying conditions of position and pressure it is probable that the flow is occasionally reversed. The superior petrosal receives several veins from the lower surface of the temporosphenoidal lobe and also a small but constant group of vessels which collect from the mucous membrane of the aditus and antrum and pass through the arch formed by the superior semicircular canal to emerge from the bone in the subarcuate fossa on the posterior surface of the petrous pyramid. In the infant this fossa is as big as the internal auditory meatus and transmits a large vein. This gradually lessens in size, till in the adult it is barely visible. The superior and inferior petrosal sinuses also receive some large cerebellar veins, which come from between the superior semilunar lobule and the posterior part of the quadrangular lobule.

In the course of a middle ear suppuration pathogenic organisms may pass along the subarcuate vessels into the superior petrosal sinus and thence into the thin walled cerebral and cerebellar tributaries. From here they may pass into the subarachnoid space or even into the brain substance itself. A case has been described by Fraser,[4] in which infection passing along the subarcuate vessels was demonstrated microscopically.

It is interesting to note from the records available that out of 67 cases of brain abscess, of which 43 were in the temporosphenoidal lobe and 24 in the cerebellum, sinus thrombosis was associated in 16 and perisinus abscess in 4. In the 43

temporosphenoidal abscess cases thrombosis was present in only four and perisinus abscess in only one. It is to be noted also that in one of these the brain abscess was on the opposite side from the ear lesion. In the 24 cerebellar cases thrombosis was associated in 12 instances, while in three others there was a perisinus abscess without thrombosis. This rather points to the probability of venous embolism as an etiologic factor in the formation of abscess, in the cerebellum at any rate.

FREQUENCY OF COMPLICATIONS.

According to Milligan,[5] in acute cases extradural abscess or sinus thrombosis is the route by which infection reaches the cerebellum. In chronic cases the abscess is generally secondary to labyrinthitis.

As far as one may generalize from the numbers before me, temporosphenoidal abscess is less frequently accompanied by other complications than cerebellar abscess. There were seventeen cases of temporosphenoidal abscess without other complications. There were five in which more than one abscess was present, and included in these five are two where cerebellar abscess was present in addition. Eleven cases had some other complication, such as extradural abscess, sinus thrombosis, or meningitis Cerebellar abscess unaccompanied by other conditions was found in only three cases, multiple abscess in three cases, including the two already mentioned with temporosphenoidal abscess in addition. Cerebellar abscess and labyrinthitis occurred together three times, and cerebellar abscess with extradural abscess, sinus thrombosis or meningitis in eight cases. There was one case of subdural cerebellar abscess with extradural abscess.

From the foregoing it will be seen that in roughly half of the temporosphenoidal cases the abscess occurred without other complications. In cerebellar abscess, on the contrary, only three out of seventeen were found without other complications.

PATHOLOGY.

When I was in France I had the opportunity or carrying out some 72 postmortem examinations on head cases. With the exception of a few cases of cerebrospinal meningitis, these were all gunshot wounds of the brain. The brain lesions seen

bore a great resemblance to the otogenic brain abscesses of civil life. In almost every case there was some attempt at the formation of adhesions at the edge of the opening in the dura. In the most recent cases there was of course a great deal of damage to the brain for some distance round the actual track of the projectile. In cases which had lived for several days or longer a great deal of the damaged brain tissue had sloughed out, leaving an abscess cavity. This at first was irregular in outline, but older cases showed the formation of a fairly well marked capsule with smooth lining walls, showing a great tendency for the limitation and ultimate shutting off of the damaged area.

In the case of otogenic brain abscess the first stage is a localized patch of red softening or encephalitis. This later breaks down and forms an abscess cavity. In many cases there is a distinct tendency for the limitation of the spread of the infection and a more or less encapsulated abscess results. In other cases the encephalitis extends rapidly and causes death from increase in the intracranial pressure. An encysted abscess which had been more or less latent may suddenly become surrounded by a zone of acute encephalitis with subsequent secondary abscess formation, as is shown by Macewen's case XXX, in which the encapsulated abscess was found inside a more recent surrounding abscess.[6]

INJURY AS AN ETIOLOGIC FACTOR.

Five of the cases under review had received some injury to the head sufficient to cause loss of consciousness. This appeared to play some part in the formation of the abscess, as the symptoms dated from the injury in most of them. It is only reasonable to suppose that when an injury occurs during the course of a middle ear suppuration new avenues of infection may be opened up.

With regard to the question of trauma, the mastoid operation must also be considered. In seven cases a mastoid operation had been done with apparently no suspicion of the presence of a brain abscess. After a period varying from some hours to a month symptoms of brain abscess began to show themselves. If one asks why, he is faced with three alternatives: did the trauma of the mastoid operation cause the brain

abscess, or did it light up an already existing latent abscess, or had it nothing to do with it? These questions are difficult if not impossible to settle from the small number of cases available for study. On the other hand, in several instances patients were seen with symptoms leading to suspicion of an intracranial complication. These disappeared entirely after operation for one or two weeks, only to reappear along with other more definite signs. The question then must be left open for the present. The latent period after mastoid operation requires investigation, as it is obvious from numerous cases that the abscess was already present at the time of operation, yet all the suspicious symptoms disappeared for a while, leading the surgeon to think he had been mistaken.

CAUSE OF DEATH.

In a study of 25 fatal cases of brain abscess in the available literature it was seen that diffuse purulent meningitis was the cause of death in twelve. In two of these the meningitis had been set up by leakage from the abscess into the subarachnoid space. Five cases died from pressure symptoms, in three of which death was due to acute encephalitis or edema. In three cases the patients died with pressure symptoms due to a second undiscovered abscess. In three death was due to rupture of the abscess into one of the ventricles. In my own experience this latter complication is generally fatal within a few hours, but in one of Fraser's cases death did not occur till 17 days after rupture.

GENERAL SYMPTOMS OF BRAIN ABSCESS.

It must be clearly understood that there is no one symptom or sign which is constantly present. By analyzing a large series of cases a fairly definite composite picture may be formed, but it is unusual to get more than a very small proportion of the symptoms and signs in any one case.

Brain abscess is generally described as showing three clinical phases: (1) An initial, (2) a middle, and (3) a terminal stage. The first stage is very seldom seen in hospital. It corresponds with the formation of an area of red softening and is generally ushered in by a chill, headache and vomiting. The chill may be merely a feeling of cold with cutis anserina, or may

be an actual rigor. The temperature may be slightly raised. In the middle stage the signs of increased pressure predominate. The senses are blunted, the cerebration is slow and the mind is dull. Headache is severe but does not cause outcry. The pulse and respiration rates are slow, and the temperature is normal or subnormal. The terminal stage is short and is characterized by coma, incontinence and increasing respiratory failure. Hyperpyrexia and rapid pulse may immediately precede death.

Encephalitis, abscess and meningitis give a series of symptoms which are common to each, but varying in degree. The symptoms are in the main due to the accompanying increased intracranial pressure. These are headache, vomiting, mental dullness, drowsiness, optic neuritis, slow pulse and often subnormal temperature. In many of Macewen's cases the onset of symptoms was ushered in by a rigor or feeling of cold (ten out of nineteen). However, in four of them there was sinus thrombosis, and in several others there was a perisinus abscess with a suspicion of involvement of the sinus. In the forty-seven other cases here analyzed rigor or chilly feeling was only mentioned five times, and in three of these there was sinus thrombosis. That is, out of sixty-six cases in all, rigors occurred in fifteen, and of these fifteen, nine were cases with sinus thrombosis. From this it would appear that rigors or chills sometimes occur with simple brain abscess, but that in most cases where such a symptom is noted it is due to an accompanying sinus thrombosis.

Headache is one of the most constant symptoms and was present in fifty cases. In some of the remaining cases its presence or absence could not be ascertained, as the patient was already comatose. Where details were given the headache had usually commenced on the affected side and was extremely acute. In the initial stage it was noted in several cases as being so severe as to prevent sleep. Later when the patient was passing into the second stage it was generally frontal or occipital, or both. The headache of abscess is extremely severe, with exacerbations at times. It does not, however, as a rule cause the patient to cry out as it does in meningitis, where the headache is excruciating. This is so, even with the socalled serous meningitis. Aboulker[7] emphasizes

the difference between the headache of abscess and that of meningitis. The patient with abscess, when interrogated, will tell you that he has a terrible headache, but he does not, as a rule, volunteer the information.

Mental disturbances manifested in several ways have been described by various observers, as slowness of cerebration, obnubilation or clouding of the intellect, apparent stupidity, absent?mindedness, lack of attention, psychic depression, etc. In several instances noted the mental condition was practically the only symptom of the abscess. In one case reported by Aboulker a girl, aged 15, had been attending a clinic with chronic middle ear suppuration. It was noticed that she was easily fatigued by questions and was very absent minded. On the strength of this, cerebral abscess was diagnosed, although there was no headache or any other sign, and lumbar puncture was normal. The patient, however, refused to go into the hospital, but returned a month later with mastoiditis. An abscess was found in the temporosphenoidal lobe and another one in the cerebellum. Voisin[8] reports the case of a soldier who was thought by his comrades to be feeble minded. He was admitted to the hospital with acute middle ear suppuration, and he also had an abscess in the right tonsil, with some delirium. Some days later there was a little transitory mental confusion; ten days later he suddenly developed torpor and hemiplegia and died in 48 hours. At the postmortem an abscess the size of an egg was found in the left ascending parietal region. Another was forming in the occipital lobe. He had never complained of headache or any other sign. Adson[9] notes symptoms of mental disturbance in fifteen of his twenty-six cases. They were lack of attention, loss of memory, delirium and coma. This clouding of the intellect, inability for sustained attention, and difficulty in exerting one's strength are due to the increased intracranial pressure, and also to the presence of a large amount of toxin in the cerebrospinal fluid. As a rule these are not noticed till the second stage, when signs of drowsiness predominate.

Drowsiness is not found in the early stages, as a rule, but only in the second and final stages. As the intracranial pressure increases, so does somnolence till it merges into coma. In the second stage the patient spends most of his time lying

half asleep. He can be roused and will answer questions, but immediately drops off to sleep again.

Vomiting occurs in the early and middle stages, but is rarer in the last stage. In the cases under review it was noted as a symptom thirty times. However, it was doubtless present in many more of the cases, as in those which were carefully detailed it was always there. The characteristic of vomiting due to brain abscess is that it bears very little relation to the taking of food. As far as the act of vomiting itself is concerned, it is like that from any other cause, but is usually unaccompanied by nausea. Projectile vomiting, socalled, is not very common. It was noted as such in only one case. The vomiting in the initial stages of brain abscess and in sinus thrombosis and meningitis frequently accompanies a rigor. Moving the patient in bed during an examination very often brings it about.

Nausea, which is so marked a feature of labyrinthitis, is generally absent in cerebral abscess, though sometimes present in cerebellar abscess.

Obstinate constipation is a frequent symptom when the intracranial pressure becomes great. The tongue is also often very thickly furred.

Optic Neuritis.—Adson (loc. cit.) found choked discs in nine out of his twenty-six cases. Logan Turner (loc. cit.), in fourteen cases of intracranial complications of otitis media, found optic neuritis in none of them, though in some there was blurring of the edges of the discs and engorgement of the vessels. The condition of the eye grounds was noted in thirty-four cases in this series. Double optic neuritis occurred in twelve, and in three of these it was more marked on the affected side, while in one it was more marked on the opposite side. In seven cases there was optic neuritis on one side only. In six of these it was on the affected side, and in one on the opposite side. There was blurring of the discs in four cases, and engorgement of the vessels in four cases, in one of which it was more marked on the opposite side. The discs were normal in eight cases. That is to say, in twenty-six of the thirty-four cases there was a positive finding, and the condition was generally more marked on the affected side. Optic neuritis is not, as a rule, seen in cases where the in-

crease of intracranial pressure had lasted less than a fortnight. In two of the cases under review there was no neuritis when first examined, but it developed some time later. In a case which I saw some years ago, after repeated examinations neuritis was seen to develop only two weeks after the onset of symptoms. Optic neuritis appears earlier and is more severe in posterior fossa infections than in those of the middle or anterior fossa. Blindness is also said to be much commoner as a result of cerebellar than of temporosphenoidal abscess. According to Macewen, the optic neuritis is rarely followed by atrophy, and the sight generally returns when the brain condition is relieved. From the foregoing it will be seen that the absence of optic neuritis does not rule out the diagnosis of brain abscess, but its presence is an important confirmatory sign.

Pupils.—In the early irritative stages the pupils are contracted and active. When pressure symptoms begin to predominate they tend to dilate and are sluggish in their reaction to light. The state of the pupils was noted in twenty-one cases. In five of these, both pupils were dilated and fixed, and in all of them other pressure symptoms were very marked. In six they were equal and small, and in all of these except one pressure signs were not severe. The mental state was rather irritable than dull. In the one case, a fatal one with double cerebellar abscess, the pupils became contracted just before death. In ten instances the pupil on the affected side was dilated and in most of them it was fixed. In one of the cases the pupil was active and the patient was restless. In the others the patients were dull and showing signs of severe pressure. Macewen states that when the abscess is in the frontal or temporosphenoidal lobe the pupils may be either small or large, but especially sluggish. Sometimes the only difference is that the pupil on the affected side is more sluggish. In diffuse meningitis the pupils are usually equal and contracted till a late stage, when they dilate.

Pulse.—In the initial stage of brain abscess the pulse may be frequent, but in the stage when cases are usually seen the pulse is generally slow. A pulse rate of 50 to 70 is very common. In this series the condition of the pulse was noted in forty cases. In four of them the lowest readings were be-

tween 40 and 49, in ten between 50 and 59, in eleven between 60 and 69, in none between 70 and 79, in two between 80 and 89, in four between 90 and 100 and over, one was noted as being normal and six were noted as subnormal. In the group over 90, one was a child of 8, who died. The other three were uncomplicated cases in adults who recovered. The four cases with a pulse rate between 40 and 49 were also uncomplicated. From the above figures it would appear that the tendency is to remain subnormal and that accompanying complications or intercurrent disease do not have any marked effect on this tendency. When an abscess ruptures, either on to the surface of the brain or into the ventricles, there is generally a marked acceleration before death, as is shown by the case of Fraser's (loc. cit.), where the pulse rose to 120 and temperature to 104, and another in which the pulse was 130 and temperature 104, and in Macewen's case XXX, where the pulse was 140 and temperature 105.8.

Temperature.—It is more difficult to generalize on the question of temperature, as it is much more variable than the pulse, but in a large number of uncomplicated cases it tends to be subnormal. When other pressure signs are present, 97° F. is common. However, when meningitis exists as well, the temperature may remain above normal, and the signs of abscess may be completely masked. Meningitis is characterized by a high temperature, with a normal or subnormal pulse rate. As already noted in connection with the pulse, after rupture of an abscess into the ventricle, hyperpyrexia is the rule. I have seen a case with temperature of 107 just before death.

Respiration.—The respiration rate is generally about normal with a tendency to slowing. When the intracranial pressure is becoming severe and the patient is becoming comatose, Cheyne-Stokes respiration or other intermittent breathing is common. Intermissions between breaths may last half a minute. When death from increased pressure is approaching, the respiratory function is usually the first to go. Patients can be kept alive for hours if artificial respiration is carried out. During operation there is very frequently cessation of breathing, necessitating artificial respiration till the abscess is opened. This is particularly so in cerebellar cases.

Leucocyte Count.—Darling has studied the blood picture in a large series of ear cases and the results of his investigations are quoted here.[10] In acute middle ear suppuration with mastoiditis the average white blood count was 12,400 per cub. mm.; maximum, 20,600; minimum, 7,200. In chronic middle ear suppuration the average was 10,200. In chronic middle ear suppuration with mastoiditis the average was 17,600; maximum, 22,100; minimum, 12,700. General anesthesia, particularly with chloroform and ether, causes an increase in the leucocytes of about 8,000 for a period averaging three and one-half days. In his series there were seventeen intracranial cases. In extradural abscess the average was 18,400; maximum, 30,000; minimum, 9,300. In six brain abscesses the average was 12,500; maximum, 14,000; minimum, 10,300. In meningitis the average was 25,500; maximum, 33,000; minimum, 17,000. Sinus thrombosis averaged 14,500, but, leaving out one atypical case, it was 17,000. Darling states that if the polymorphs are under 77 per cent, probably no intracranial complication is present. Single examinations are not so useful as repeated ones, as it is only then that the change in the blood picture gives valuable information. Adson gives an average of 16,000 in brain abscess cases. In some instances the count was normal.

From the foregoing it is evident that the leucocyte count does not help materially in the diagnosis of brain abscess. In obscure cases, in which one is uncertain as to the presence or absence of a latent abscess, the mastoid condition alone would give a blood picture very similar to that of brain abscess. However, as one can exclude extradural abscess and meningitis by other means, and if the uncertainty occurs more than five days after operation or in the absence of acute mastoiditis, a leucocyte count of 12,000 to 16,000 would suggest the presence of abscess.

Cerebrospinal Fluid.—Adson states that when there is no diffuse encephalitis or meningitis the cell count in the cerebrospinal fluid is normal in brain abscess (2 to 5 lymphocytes per cub. mm.). In cases complicated with meningitis it may be as high as 108,000. Fleischmann[11] in an extensive series of examinations of the cerebrospinal fluid in ear cases had twelve of brain abscess, of which seven were otogenic. There

were twenty-three lumbar punctures in all. He found the pressure increased in most of the cases, though not in all. There was no increase of the cells in three, but in ten there was a marked increase of lymphocytes up to between 30 and 300. The total albumin and globulin was increased, though not constantly nor to a marked degree, the normal albumin content being about 0.2 to 0.5 per cent. Sugar is present in brain abscess cases, but is generally absent in meningitis. The change in the number of cells is the most constant sign.

Lumbar puncture had been performed in twenty-four of the cases in this series one or more times. The results were so variable and the points noted and omitted so different that one cannot draw any very definite conclusions, except that the pressure was in most cases raised. In those cases where the cell count was mentioned it was increased, and in quite a number the increase was enough to cause turbidity, yet no organisms could be seen on direct smears, and cultures remained sterile. In what the French writers describe as méningite puriforme aseptique a similar condition is found, and Aboulker (loc. cit.) states that in it the symptoms are much more alarming than in the much more serious brain abscess. The headache is much more intense, etc.

In other cases the fluid was clear, but contained cocci of various sorts. While the presence of germs in the cerebrospinal fluid must always be regarded as an ominous sign, these cases are by no means always fatal, as there were four in this series alone which recovered.

In a case of temporosphenoidal abscess reported by Fournioux,[12] in which meningitic symptoms were present, the first lumbar puncture showed nothing abnormal. After a radical mastoid operation the symptoms subsided. Nine days later there were again meningitic symptoms and a brain abscess was opened. Next day the cerebrospinal fluid was turbid, but the pressure was not increased. There was a heavy deposit with 80 per cent polymorphs, 15 per cent lymphocytes, and 5 per cent endothelial cells. Albumin was present; no glucose; some cocci but culture negative. Four days later cerebrospinal fluid unchanged. Eight days later, flocculent deposit; polymorps 70 per cent; albumin increased; sugar absent; germs present.

Twelve days later a trace of sugar present; lymphocytes 90 per cent. Recovery uneventful.

So much for the general symptoms and signs of brain abscess. Let us now consider the symptoms and signs which help us to determine the site of the abscess. They comprise to a great extent irritative and paralytic phenomena, but also disturbances of speech and other mental functions in the case of the cerebrum, and troubles in coordination, balance, etc., in the case of the cerebellum.

Localized Pain.—Macewen draws attention to the value of percussion of the head in locating the site of an abscess. Patients with brain abscess generally resent tapping over the region of the abscess, while they do not mind it over the rest of the skull. Adson states that he found local tenderness in twelve of his twenty-six cases; in four of these it was not over the site of the abscess, but in the remaining eight it was a confirmatory sign. In several of the other cases reviewed here tenderness to percussion was noted.

TEMPOROSPHENOIDAL ABSCESS.

In addition to the general symptoms mentioned above, temporosphenoidal abscess often shows some localizing signs, such as paralysis on the opposite side, convulsions, aphasia, etc.

Paralysis.—In forty-three cases of temporosphenoidal abscess hemiplegia of the oposite side was noted in seven, and weakness of the opposite side in three. The oculomotor nerve of the same side was involved in five cases. In four of these there was ptosis, and in the other case the whole of the nerve was paralyzed except for the branch to the levator palpebræ. In one case the sixth nerve of the same side, and in one that of the opposite side was paralyzed.

Paralyses are less common in cerebellar abscesses, but when paralysis or paresis is present it is on the same side as the abscess, in contradistinction to temporosphenoidal abscess. Weakness of the arm of the same side was noted in three cases only. In one of these the paralysis of the limb was complete. but function was restored after the abscess was opened. There was paralysis of the sixth nerve on the same side in two cerebellar cases and of the oculomotor in one. From this it might appear that temporosphenoidal abscess shows more localizing

signs than cerebellar abscess. This is, however, not the case, as the cerebellar localizing signs are of a different category and will be discussed later.

The paralysis or paresis of the limbs of the opposite sides in temporosphenoidal abscess hardly require comment, except that if present it is important to note the order of involvement of the limbs. If face, arm, leg is the order of involvement the chances are that we are dealing with a subdural abscess or meningitis. If in the opposite order it is more likely to be pressure on the internal capsule. If the infective process extends as far as the basal ganglia, part of the lenticular or thalamic syndrome of Déjerine and Roussy may be met with. This syndrome consists of hemiataxia with marked disturbances of deep sensation with slight hemiparesis. Hemichorea and hemiathetosis may be associated. Macewen's case XXIX showed paralysis of the arm and leg of the opposite side, accompanied by choreic movements. Paralysis of the third or sixth nerve of the same side may be due to pressure on the nerve trunks or involvement of the peduncle or basal meningitis.

Paralysis of the cranial nerves is more often due to basal meningitis than to brain abscess. Paralysis of the sixth nerve on the affected side occurs not infrequently as a result of localized pachymeningitis at the apex of the petrous bone with headache and a few other symptoms, and this is known as the syndrome of Gradenigo. It is not usually fatal but only transitory.

Reflexes.—The examination of the reflexes as a rule gives very little help in the diagnosis of brain abscess. In temporosphenoidal abscess the knee jerks are sometimes altered on the opposite side, but generally only when there are already paralytic phenomena. In cerebellar abscess the knee jerks are usually unaltered.

Convulsions, etc.—Adson reports Jacksonian epileptic attacks in twelve and grand mal epilepsy in four of his twenty-six cases. In the series studied here, convulsive movements of the limbs were noted in eight of the sixty-seven cases. Three of these had repeated generalized convulsions, and in two of the three the patients had meningitis as well. Jacksonian fits or twitching of the limbs on the opposite side occurred in three

cases. Seven of the eight cases were temporosphenoidal abscess. The remaining case was one of cerebellar abscess complicated by basal meningitis. Apparently, then, convulsions or jerkings are not very common in uncomplicated brain abscess, but when present are of considerable value in localizing the condition. Twitchings are rare in cerebellar abscess.

Disturbances of Speech.—The modern conceptions of aphasia advanced by Pierre Marie have been fully substantiated, according to Chatelin,[13] by the study of gunshot wounds of the brain occurring during the great war. Briefly, Marie distinguishes between aphasia proper and motor aphasia, which he terms anarthria or dysarthria, according to the degree of the disability. In the latter condition the comprehension of speech is retained. Aphasia is the loss of understanding of speech, and may consist in a difficulty in finding words to name objects or convey ideas, using wrong words (paraphasia) or, on the other hand, there may be a lack of comprehension of words heard. In alexia, or visual aphasia, or psychic blindness, there is a lack of comprehension of things seen. There is always more or less clouding of the intellect associated with these different forms.

Anarthria is associated with the lenticular zone of Pierre Marie, which is defined as including the central gray nuclei, the internal and external capsules, the insula, the motor cortex and some of the important white fibers. The third frontal convolution or area of Broca is not included. Anarthria is not connected entirely with the left side of the brain, but is also found in corresponding lesions of the right side.

True aphasia is associated exclusively with the left side of the brain in right handed individuals. It is located in the area of Wernicke, which includes the gyrus supramarginalis, gyrus angularis, the posterior end of the two first temporal convolutions and the subjacent white matter as far as the wall of the descending horn of the lateral ventricle, which immediately adjoins the already mentioned lenticular zone.

Lesions in the motor area, namely, ascending frontal convolution, produce anarthria with or without accompanying paralyses. Lesions of the left temporoparietal region in the area of the supramarginal gyrus produce aphasia with some anarthria. Lesions in the middle and posterior parts of the

temporal convolutions produce a difficulty in naming objects. The intellectual faculties are very much affected, especially that of calculation. There is very little anarthria. A lesion in the left posterior temporal and angular convolutions produces alexia predominantly, frequently associated with hemianopsia if the lesion is deep. Dysarthria or difficulty of speech may also be caused by cerebellar or bulbar lesions.

In thirty-one temporosphenoidal abscesses there were fifteen on the right side, thirteen on the left side, and in three instances the side was not stated. Aphasia was noted in seven of the thirteen. Macewen's patient XX, who had an extensive necrosis of the left temporosphenoidal lobe, could not understand speech properly and mistook the meaning. There was paraphasia, or use of the wrong words, and considerable mental obscuration as well. In another case (Fraser) a child could name some objects but not others. She could name knife but could not name keys, though she said they were for opening doors. In another case (Macewen, XXXVII), where there was a cerebellar abscess and at the same time a large subdural abscess over the first and second temporal convolutions, there was complete word deafness. The patient only arrived at the meaning by repeated guessing, but he had complete command of language to answer. Later, the same patient developed visual aphasia, or psychic blindness. He could not recognize objects held up to him till he touched them. In one case of cerebellar abscess "motor aphasia" was noted. This was probably dysarthria connected with ataxia and other disturbances of coordination.

CEREBELLAR ABSCESS.

Cerebellar abscess is much richer in localizing symptoms and signs than temporosphenoidal abscess. Among the symptoms and signs are nystagmus, vertigo, troubles of balancing and coordination, such as falling, pointing error, ataxia, lack of muscular tone on the affected side, intention tremor, etc. Repeated yawning and rigidity of the masseters were frequently observed by Macewen.

Nystagmus, apart from that of ocular origin, which is easily distinguished, is almost invariably due to a lesion of an irritative or destructive character, either in the labyrinth or in the

posterior cranial fossa. The commonest cause of labyrinthine irritation is an erosion of the labyrinth wall, generally in the region of the external semicircular canal. The labyrinth is functionally intact and the condition is easily diagnosed. The nystagmus and giddiness are only occasional and are generally produced by sudden movements of the head. The detailed examination need not be discussed here. Destructive lesions of one labyrinth cause violent dizziness and nystagmus, accompanied by nausea and vomiting. The nystagmus of destructive lesions of the labyrinth is invariably directed away from the affected side—that is, the slow or labyrinthine movement is towards the affected side, and the quick or cerebral movement towards the opposite side. The nystagmus is mixed in character—i. e., rotatory and horizontal. Labyrinthine nystagmus is most marked at first, but gradually diminishes from day to day and finally disappears. The labyrinth does not respond to stimulation by the caloric or rotation tests and is absolutely deaf. Associated with nystagmus to the opposite side is a tendency to fall to the diseased side, or to deviate towards the diseased side in walking. Romberg's test gives falling to the diseased side. If the head is turned away from the diseased side the patient falls forwards; if turned towards the diseased side he falls backwards. Barany's pointing test gives deviation towards the diseased side with one or both arms.

Cerebellar nystagmus differs in several important respects from labyrinthine nystagmus. In the first place, it is as a rule not continuous. One day it may be present and another day absent. Its direction also may vary from time to time; one day it may be directed to the right and another day to the left. In a case of Fraser's[14] there was spontaneous nystagmus one day to the sound side of the first degree; next day there was nystagmus of the second degree to the affected side. Nystagmus due to cerebellar lesions does not rapidly disappear as does labyrinthine, but on the contrary lasts a long time and tends to increase. In cerebellar lesions there is a tendency to fall or deviate to the affected side. This falling tendency is uninfluenced by altering the position of the head, and according to Holmes[25] is unaffected by closing the eyes. In cerebellar lesions the pointing test generally shows a deviation of

the arm on the affected side, and the direction of the deviation has no relation to the direction of the nystagmus if the latter is present. The deviation is generally towards the affected side. Gordon Holmes in his observations on war wounds of the cerebellum has done much to systematize the symptoms and signs. He states that nystagmus is almost invariably present and points out a further characteristic of cerebellar nystagmus. "It is essentially a fixation nystagmus—that is, it can be seen as a rule only when the patient fixes an object." There is a rest point towards which the eyes tend to deviate when the patient is at rest. This point is usually 10 to 30 degrees to the unaffected side of the middle line. When the patient is asked to look at an object straight in front of him, the eyes tend to roll slowly to the rest point on the unaffected side, and are brought back by quick jerks. On looking towards the affected side the nystagmus becomes much more marked.

Labyrinthine nystagmus, on the other hand, is best seen when the patient does not fix an object. On fixation the nystagmus may be controlled and may disappear.

Loss of muscular tone is a constant feature of cerebellar lesions. This is evidenced by the unnatural attitudes assumed by the limbs on the affected side. If the limbs are passively moved the affected ones flop about much more than those of the other side. The knee jerks are unchanged.

There is delay in initiating and stopping any voluntary movement. This is brought out by asking the patient to perform rapidly alternating movements with both hands simultaneously. The limb on the affected side lags behind (adiadochokinesis).

Disturbances of coordination when analyzed are resolved into (1) asynergia, or defective synchronization of the muscles, resulting in intention tremor; (2) dysmetria, or failure to gauge the force and extent of a desired movement.

There are many other signs deserving mention, but time and space do not allow of it at present. For the same reason the significance and interpretation of the vestibular reactions cannot be discussed, except to state that the condition of the labyrinth must in all suspicious cases be thoroughly investigated. As already mentioned, cerebellar abscess is very frequently associated with labyrinthitis, and merely to discover

that one labyrinth is not functioning is not enough . The vestibular reactions must be analyzed to ascertain whether there is any cerebellar lesion as well.

REPORT OF A CASE OF CEREBELLAR ENCEPHALITIS.

A boy, aged 17, was seen on April 15, 1921, with the following history. His left ear had been running all his life. He had had attention for this from time to time, and the condition had improved. At Easter he had had a knockout blow while boxing and had not been quite well since then. Five days ago he had been out in a cold wind and soon afterwards began to have some pain in the left ear, which soon became very acute, and the discharge became very profuse. He remained several days in the school sickroom and came home on the 14th. On the journey home in the train he suffered from an extremely severe headache on the top of his head, with great dizziness and vomiting. Since arriving home had vomited several times and had some chilly feelings. The headache was still present on the top of the head and had been constant for two days.

On examination there was copious purulent discharge from the left ear with distinct mastoid tenderness. The whole left drum membrane was gone; right drum membrane normal; very deaf in left ear; could hear raised voice with noisebox in right ear; temperature 101.5 F. It was higher the night before. Nystagmus absent; pointing test normal; no adiadochokinesis.

Operation: Cortex thin; bone extensively softened and extremely vascular; cells not numerous, but some pus in them; antrum also contained pus; radical operation completed; Körner flap; eustachian tube curetted; no erosion of tegmen; sinus far back; exposed deliberately on account of high temperature, headache and chills; found healthy; wound packed and closed.

April 16. Temperature 99.4. No vomiting. Headache gone.

April 17. Temperature normal. Some pain in stomach; outer dressing changed, as it was soaked through.

April 30. Doing well; posterior wound was a little septic, but now healed; cavity clean; leaving hospital.

May 2. Had a bad headache today, which began last night; was a little chilly in the evening; no vomiting; ear clean; pulse 68; temperature subnormal; complained of blurred vision; very slight barely perceptible nystagmus to left (affected side). In the evening, pulse 76, temperature 98; no neck stiffness, no Kernig; arms very weak; hand grips weak but equal; can touch nose equally with both; abdominal reflexes normal; knee jerks active and equal; no clonus; plantar reflexes not elicited; bright mentally.

May 3. Vomited several times during night; constipation in spite of two purgatives; headache worse; felt behind eyes and back of neck; marked horizontal nystagmus to left. Dr. Courtenay examined eyes and reported blurring of the left disc and congestion of vessels; pupils equal and react actively to light and accommodation; no diplopia; slight slowness of left arm in rapid alternating movements; no Kernig; pulse 80, temperature 98.4. Cold peroxid in the ear during dressing produced nystagmus to opposite side and gave sensation of rotation. Readmitted to hospital.

Evening examination: Inclined to be drowsy all day and lies with eyes shut; no mental disturbance; rest position of eyes a little to right of middle line; a little horizontal nystagmus on extreme deviation to right; horizontal nystagmus to left. Pointing test with right arm perfect at shoulder, elbow, wrist and finger from above downwards; left arm fairly good but slight tendency to deviate to left from wrist; both grips equally weak; flaccidity very marked, both sides; does not touch nose very well with forefinger, slightly worse with left; no intention tremor nor misjudging of distance; some adiadochokinesis of left arm; on standing tends to fall backwards; unaffected by turning head. Cerebellar abscess was diagnosed, but it was decided to observe the case a little longer before operating.

May 4. Condition unchanged; lumbar puncture; fluid under considerable pressure; clear; report showed globulin not increased, 70 lymphocytes per cub. mm.; Fehling reduced.

May 5. Slept badly; headache rather worse; objective signs as before. At 6 p. m. had vomited four times since morning; headache much worse; great pain on tapping over left occipital region; decided to operate now. Operation 9:45 p. m.

Nitrous oxid anesthesia; posterior incision opened up; mastoid cavity healthy; bone extremely vascular; sinus uncovered freely; profuse hemorrhage with every bite of bone; cerebellar dura uncovered in front of sinus as far as possible without opening posterior semicircular canal, and also for some distance behind sinus, where mastoid emissary vein gave a lot of trouble; dura opened behind sinus, and brain immediately bulged out; cerebellum explored in a forward direction and sensation of going into a cavity felt, but no pus found. After operation patient said head felt better; nystagmus not so marked.

May 6. Vitality low today; no more vomiting; nystagmus less; some external squint, which is not paralytic, as both eyes can move. Headache still severe; patient apathetic and lies with eyes half open; in evening Cheyne-Stokes respiration; Dr. Sims saw patient in consultation; dura opened more widely behind and also in front of sinus; brain explored again; again sensation of going into cavity, but no pus; firm brain felt beyond; condition evidently localized encephalitis; hypertonic saline dressing; 10 cc. antistreptococcus serum given.

May 7. Still apathetic, but talks sensibly, though speech rather slurred; temperature subnormal, 97.3 during night; pulse 80; respirations 20; given 50 cc. antistreptococcus serum; in evening Cheyne-Stokes respiration very marked; came on fairly suddenly at 10 p. m. Intermissions of 30 seconds in breathing; given strychnin 1/30.

May 8. Had very good night and took fair amount of nourishment; at 7 a. m. breathing suddenly became intermittent; given strychnin and camphor; seen by me 10 minutes later; had revived somewhat, pulse and respirations better; forceps put on lateral sinus, which was divided, and dura opened widely; cerebellum bulged out; patient protested vigorously; he was quite conscious, though extremely apathetic and drowsy; squint as before; given 50 cc. serum; at 6 p. m. patient quite lively and asking questions; pulse now 102; temperature 98; breathing regular and natural for first time in days; taking lots of nourishment; beginning to object to nutrient enemas today; salt packs abstracting considerable fluid from wound.

May 9. Slept naturally during night; respirations more normal; patient much more lively mentally than even yester-

day; objects greatly to dressings; very restless all day, and moving arms and legs about a great deal; still diplopia, though eyes nearly parallel; still nystagmus to left; left arm very flabby; right arm firm; both calves equally firm; left hand flops distinctly more than right on passive movement; pulse 88; still frontal headache; 50 cc. serum given.

May 10. Had a good night; brain pulsating well; forceps removed from dura; some odor from wound, probably from sloughs caused by forceps; temperature 100; still marked nystagmus to left; still diplopia, though squint not apparent.

May 11. Brain protrusion somewhat less; pulse and temperature normal.

May 12. Headache gone in evening.

May 13. Temperature dropped to 96.2 in early morning, but rose again; no headache; can move left hand fairly well with only a little intention tremor.

May 14. No headache; pulse and temperature normal; still some diplopia; brain protrusion same size, but firmer. Pointing tests: Pointing with left arm downwards from shoulder he deviated outwards, other joint movements normal; rest point of eyes now to left to middle line.

May 18. Progress good; feels well; no headache, but had a little the last two days. Brain bulging a little more; temperature 97. No diplopia; still nystagmus to both sides; white blood count 13,000.

May 20. Wound granulating well; salt pack discontinued; posterior pedunculated part of hernia about size of pigeon's egg.

May 23. Brain protrusion much smaller today; opening seen in anterior part, as though small abscess had burst. Brain pulsates better; pointing tests normal with both hands; touching nose not quite so good with left hand as with right; pulse and temperature normal.

May 25. Eyes examined by Dr. Courtenay; fundi absolutely normal; no engorgement of vessels; wasting and flabbiness of left arm now marked; adiadochokinesis marked.

June 2. Been sitting up for some days; hernia smaller; white blood count 7,000; grips of both hands much stronger; left apparently as strong as right.

June 16. Gets some headache on sitting up; hernia varies in size; pulse and temperature normal.

June 18. Sharp pain in head on sitting up; functional examination as before; white blood count 6,800.

June 21. Pulse and temperature normal; headache thought to be due to dragging of hernia on brain; hernia therefore pushed in with gentle pressure; dressing applied tightly.

June 24. No more headache or pain since 21st. Getting up.

June 27. Plastic operation under gas; flaps freed; scar tissue removed; edges brought together.

July 2. Went home today.

Further progress was uneventful. He reported on September 14, 1921; he was perfectly well and strong. There were no motor disturbances. The ear cavity was completely epithelialized and dry and the posterior scar scarcely noticeable. He was very anxious to be allowed to play football.

This case presents many interesting features. There was a definite history of injury, which possibly had something to do with the extension of the infective process to the brain. After the onset of the mastoiditis, symptoms suggestive of intracranial complications appeared. These were the severe headache, dizziness and vomiting. He had some chills, and, as the sinus was subsequently found to be healthy, these were probably due to the brain condition. The temperature also was much higher than is found in an uncomplicated mastoid, and one fully expected to find at least an extradural abscess and sinus thrombosis. There were no definite signs of cerebellar abscess when first seen, as is shown by the absence of nystagmus and adiadochokinesis, and the normal pointing test. The entire disappearance of symptoms after the mastoid operation and the latent period of two weeks were very misleading. At the end of this period headache, vomiting and nystagmus appeared. The nystagmus was significant of cerebellar involvement in being directed to the diseased side, when functional examination showed that the labyrinth was healthy. The eye grounds on the affected side showed slight changes. There was some incoordination and pointing error on the left side, and in fact most of the signs of a cerebellar lesion were present in a slight degree. The outward deviation of the left arm pointed to the anterolateral corner of the cerebellum, according to

Barany's localization, and this is just where the area of encephalitis was found. The edema apparently increased for some time after the second operation and would undoubtedly have led to death if the dura had not been widely opened up to relieve the pressure. From that point his recovery was slow but steady. The diplopia, which was evidently due to the pressure of the swollen cerebellum on the vulnerable sixth nerve, disappeared. The functional disturbances gradually diminished, and all went well except that the hernia would not go down. Later, when the patient complained of pain in the head whenever he sat up, as there was no sign of any increased pressure, it was thought that possibly the pedunculated hernia was being held by the ring of scar tissue round about, and could not get back inside the skull on that account. This turned out to be the case, as the brain tissue was reduced into the cranium with very little trouble, and the symptoms at once disappeared. The recovery from the wasting and loss of tone were remarkably rapid.

Case 2.—F. O., age 24, came to the office January 25, 1922, with a history of discharge from left ear since the spring of 1921. He had had growths removed from the ear in childhood, but the ear had not discharged in the interval. Two days previously he began to have pain in the left ear. Since this morning he had had a bad frontal headache. There was no dizziness or vomiting. He had never had any earache or headache before. No complaints in right ear.

Examination showed left ear full of pus, with a polypus in the roof of the meatus. The right drumhead showed a chalk patch; nose normal; left tonsil very large and red, right tonsil small; nasopharynx could not be seen; hearing, whisper, right, 18 feet; conversational voice, left, 16 feet; hears whisper one foot left with noise box in right; cold syringing left produced normal reaction. Polypus was removed with local anesthesia, revealing an attic perforation.

January 26. Headache and earache went away yesterday evening, but headache began again this morning and is now frontal and occipital; no vomiting; no chills; no pain in ear today; no mastoid tenderness; some pus in the left ear; tongue slightly furred; temperature 101.4; pulse 100; slight neck stiffness; Kernig sign present; right hand grip markedly weak-

er than left, although the patient is right handed; no aphasia; knee jerks sluggish, equal; no ankle clonus; pupils moderate and react actively to light; no nystagmus.

January 27. Patient slept well; still has frontal headache; vomited after salts last night; knee jerks active and equal; plantar reflexes flexor; temperature 101; pulse 66; bowels constipated; no pain in ear; discharge almost stopped; on syringing, fluid ran into nose; no tenderness on tapping skull.

January 29. Still has headache; pulse 56 after walking to office; temperature 96.8. Patient is rather dull mentally and slow in answering questions. Objective examination as before.

January 30. Admitted to hospital; mental condition brighter; no aphasia; pupils active and equal; no nystagmus; no diplopia; muscles flabby; tongue still furred; all reflexes normal, but right knee jerk slightly more active than left; still some trace of Kernig, though less than before; no neck stiffness; no headache today; coordination perfect; touches nose with both forefingers; pointing test normal with both hands; no loss of orientation of limbs; muscle sense good; no adiadochokinesia; cold syringing left produced rotary and horizontal nystagmus to right in fifteen seconds; pointed with both hands three inches to the left; pulse variable, 74 to 80, with pressure on eyeballs 56 and 70. Dr. Minnes examined eye grounds, but found no engorgement of the vessels and no papillitis; X-ray of skull showed nothing except some blurring of the mastoid cells; lumbar puncture gave opalescent fluid; no increase in pressure; examination showed opalescence to be due to contamination with blood; Fehling reduced, culture sterile; leucocyte count, 14,600; pulse still 54.

From the patient's dull mental state, infrequent pulse, subnormal temperature, headache, weakness of the right hand, increased leucocyte count as well as the chronic middle ear suppuration, an intracranial complication was diagnosed. From the fact that the disease appeared to be mainly in the attic and that the cerebellar functional tests were normal, it was thought that the condition was in the middle fossa. It was doubtful if an extradural abscess could account entirely for the condition unless it were of large size. It was probably a brain abscess. It was decided to operate and expose the dura of the middle fossa.

Operation: Cortex very thick and hard; lateral sinus far forward, healthy; on cutting down towards the antrum pus oozed out under pressure; mastoid process sclerotic with very few cells; antrum large and full of pus and cholesteatoma; tegmen apparently intact; radical mastoid completed; spur trimmed down and ossicles removed; cavity and tube curetted; tegmen removed and pus found outside the dura, which was white and sloughy; bone removed till healthy dura exposed; dura not pulsating, but no track discovered leading through it; Körner skin flap. It was decided to do no more at present, but to observe the patient for a few days, and if there was no improvement to explore the brain.

February 1. Slept badly last night; no vomiting; no headache; pulse 60.

February 2. No change; pulse in the sixties; right hand still rather weak.

February 3. Pulse in the seventies today; no headache: eyes examined by Dr. W. G. Fraser, who reported eye grounds normal and no limitation of visual fields nor alteration in the color fields. The culture from the mastoid pus gave a weak growth of streptococci.

February 4. Wound dressed, lower end septic, cavity clean; leucocyte count 17,000; lumbar puncture gave clear fluid, with no increase in tension; subsequent report on fluid, 120 lymphocytes per cub. mm.; Fehling reduced, cultures sterile.

February 17. Not much change in the last week; no headache, no vomiting, no pain, but patient always somewhat listless and apathetic; hand grips as before. In conversation it was discovered today that he could not remember what battalion he had been in in the army nor where he had been stationed. He could not remember the name of the village where he had been born, although he knew it was near London, England. He did not remember the name of the ship in which he crossed the Atlantic while in the Army. On the other hand, he remembered exactly how long he had been in hospital. He could not name a toothpick, but said it was for cleaning the mouth, similarly with keys, penknife, and other familiar objects. Eye grounds again examined, but showed no change.

February 18. Mental condition much the same as yesterday, but aphasia much less evident; could read and write a lit-

tle, though not well, as education had been poor. No paresis of any of the cranial nerves; touch and pain felt equally well on both sides; taste and smell normal; right hand grip still weaker than left; power of legs equal; musculature poor; no loss of orientation of limbs; no astereognosis; Romberg negative; coordination perfect; leucocyte count 13,000; polymorphs 77 per cent.

February 22. Leucocyte count, 11,800; aphasia improved.

February 23. Said eyes felt funny today and that he saw double for a short time; vomited once in early morning; no headache; temperature 97.5; pulse 60. Clear mentally, but not good at finding words; no diplopia; convergence good.

February 25. Had a good day yesterday. This morning early had severe frontal headache; vomited; headache later passed off; been drowsy all morning.

February 27. No more vomiting or headache; leucocyte count, 13,000; eyes examined by Dr. Minnes, who reported retinal veins a little congested; a little diplopia at a distance, but impossible to say which muscle at fault.

It was felt from the first in this case that there was a temporosphenoidal abscess, yet after the extradural abscess had been drained his pulse and temperature returned to normal. Again, it was difficult to be sure that the paresis of the hand was a recent condition, as there was no change during the whole illness. When his wife was questioned as to his normal mental condition she stated that he had always been very quiet and silent and was rather dull. She had not noticed any change in him till within the last week. The aphasia was at times quite absent and was at no time very marked. The cerebrospinal fluid never showed any increase of pressure, although the cell count was increased. The only sign which was present throughout was the leucocytosis. However, in the last few days there had been the headache, vomiting and diplopia, so it was decided to explore the brain through the mastoid cavity.

March 1. Operation: Posterior wound opened up and cavity found to be clean and partially healed; dura of middle fossa bulging and not pulsating; incised and brain found loosely adherent; brain incised with bistoury, and at a depth of about one-eighth of an inch thin bluish pus was found;

about an ounce drained out slowly. The cavity had smooth walls and extended forwards about an inch, upwards and inwards about one-half inch, but not backwards at all. It took about 30 minutes for the cavity to empty. It was washed out very gently with saline and two tubes stitched in. The pulse and respiration were good throughout the operation. Later in the evening conscious, comfortable; pulse 90; temperature 99.

March 2. Occasional diplopia still; no headache, no vomiting; not a great deal of pus; more serous fluid.

March 4. Not so well today; looks pale; rather drowsy; pulse and temperature normal; abscess cavity not collapsed; not discharging as much as expected; in the evening pulse 69.

March 5. Restless through the night; vomited in early morning; complains of severe occipital pain; moaning and restless; temperature up to 102; dressed and thick pus found on dressings; about three-fourths of an ounce of thick pus pulsated out of brain, apparently from further forwards and further back than the original cavity; headache relieved immediately afterwards; leucocyte count, 25,200. In the afternoon he was semicomatose and groaning all afternoon; had a chill, and at 4 p. m. temperature rose to 104.2 and pulse 136. Died 5:50 p. m. He had had no convulsions.

Postmortem: Dura normal; convolutions on left parietal eminence slightly flattened; no exudate on vault; brain removed; firmly adherent round wound; some turbid exudate in interpeduncular space, none elsewhere; abscess cavity thick walled and leathery. Immediately outside the capsule there was an area where the brain was broken down and from which the thick pus had evidently come on the day of death; brain hardened before cutting. When the temporosphenoidal lobe was sliced a yellow soft area of encephalitis was seen to extend right up to the descending horn of the lateral ventricle, in which there was a little pus. There was none in the body of the lateral ventricle, though the fluid was turbid. Culture from the pus obtained at the last dressing gave staphylococcus albus.

This case presented many interesting points in diagnosis. He had had no headache except at the beginning and at the end of his illness. The only time he vomited during the earlier period of observation was after taking a strong dose of salts;

he only vomited again towards the end, when the encephalitis had begun to spread round the abscess capsule. The weakness of the right hand did not involve any of the other muscles of the limb. The aphasia was rather vague and transient, being only apparent for a day or two. As most of his symptoms gradually lessened one had considerable hesitation in exploring a brain which might have been only congested and gradually healing. However, after waiting some weeks without complete recovery it was felt that the time had come to explore. Arrangements were accordingly made to operate on the 20th of February . A neurologist was called in the day before to examine the patient, but on that day, as it happened, there was no aphasia, nor was there any other sign of brain abscess. He advised against operation, which was accordingly deferred in the meantime. However, a few days later more urgent symptoms appeared, and the operation was performed and the abscess drained. The fatal result was disappointing considering the encapsulation of the abscess, but was evidently due to the onset of encephalitis before the operation.

In conclusion, I should like to state my conviction that cases of brain abscess, though difficult to detect on account of the want of obvious symptoms, may be generally revealed by thorough and repeated neurologic examinations.

Taking the obscurity of the condition into consideration, we cannot afford to neglect any of the present diagnostic methods, but in every suspected case should subject the patient to an absolutely complete examination, including psychologic, neurologic, blood and cerebrospinal fluid examinations, etc. I would go further and state that every patient before undergoing a radical mastoid operation should have a neurologic examination.

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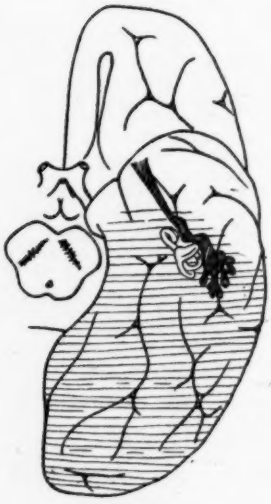

Fig. 1.

XLIII.

THE INTIMATE LYMPHATICS OF THE TRACHEA.*

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In a recently published and noteworthy series of studies on experimental lobar pneumonia by Blake and Cecil of the Army Medical School, there is among other strongly verified conclusions the statement that such pneumonia can only be produced experimentally by the direct instillation of the various types of pneumococcus into the trachea. They state that the typical lobar lesions cannot be brought on by spraying the nose and throat with virulent suspensions of pneumococci, but if the organisms are permitted access to the lower trachea, pneumonia results. The work was carried out on a large series of monkeys by injecting with sterile precautions broth suspensions through a fine needle inserted into the lumen of the trachea between the tracheal cartilages just below the larynx. The injections were made without producing coughing or gagging.

The principle involved in this work recalls a paper of Freudenthal's, published several years ago, discussing the treatment of intrathoracic conditions by intratracheal injections, in which the author offers some convincing proof that substances injected into the trachea do not pass beyond its lower limits. The same writer, in the discussion following the reading of Dr. T. W. Moore's article on intratracheal injections read before this society in 1919, said: "It is important to remember when treating the bronchi with this method that substances injected do not go down much below the level of the bifurcation."

In this connection the experiments of Coakley may also be recalled, in which after the injection of India ink solutions into the trachea of rabbits it was found after short periods of time, varying from fifteen minutes to two hours, that little or

*Candidate's thesis: American Laryngological, Rhinological and Otological Society, May, 1922.

no pigment could be found in the trachea, bronchi or alveoli, having disappeared, the author states, by lymphatic absorption.

These deductions—i. e., that suitable bacterial injections into the trachea produce lobar pneumonia,—that such injections (in doses not large enough to produce choking) probably never reach beyond the beginning of the primary bronchi,—and thirdly, that there is definite evidence of absorption in the mucous membrane of the tracheal wall,—these deductions very naturally awaken a degree of curiosity regarding the intimate lymphatics of the trachea, gratification of which is sought in vain among modern reference works. There appears to be available but few texts describing the actual lymph vessels of the wind pipe, in all instances volumes of Continental origin, and in none is the subject given more than a brief paragraph. In the divisions on the lymphatics in the larger systems of anatomy there is no mention made of the lymph vessels; text and illustrations are limited solely to a brief exposition of the tracheal lymphatic glands. The special works on the subject refer without exception to the difficulty of injecting the mucosa of the tube for determination of the distribution of the smaller vessels.

Periodical literature is devoid of any contributions to the subject, with the possible exception of the work of Winternitz, Smith and Robinson, on the rôle of the tracheal mucous membrane in the pathogenesis of pneumonia—and in this paper details are conspicuously absent.

Laryngologists seem to have neglected the matter entirely, and on account of its relation and importance to the ever increasing field of tracheobronchoscopy, I venture to bring some observations before you.

II.

In the fifth century B. C., Hippocrates referring to his discovery of a "white blood stream" and of vessels which take up nourishment from the bowel, was probably the first writer in history to mention the lymphatic system. Within three hundred years of his time we read of observations by Aristotle of peculiar vessels containing a colorless fluid, to be found

accompanying certain groups of veins and nerves. Later there is recorded similar teachings by Erasistratus, Herophilus and Galen. This fragmentary knowledge was lost during the passage of medieval ages, and it was not until the middle of the seventeenth century, when Asellius of Pavia discovered the chyle vessels in animals, that the subject stimulated early modern anatomists. Following this demonstration there was a rapid succession of discoveries. The chyle vessels were found in man; Pecquet and Van Horne dissected the thoracic duct and cysterna chyli; lymphatics in the breast were isolated, and lymphatic trunks were demonstrated individually in various parts of the body. The eighteenth century marks the beginning of our modern conception, for at this time injection of the lymphatic trunks was introduced, and it was shortly demonstrated that the chyle stream and peripheral lymphatics were parts of a single general system.

The early efforts with injections were coarse and unsatisfactory. Air, milk and various colored solutions were put into the larger lymphatic trunks under pressure, the ramifying branches being followed for varying distances. Much of our knowledge regarding the gross lymphatics results from the injection of mercury, a method introduced by Anton Nuck and used consistently by anatomists for a long period. The method was a difficult one, however, and only successful in the hands of a few trained men. The technic for which Fohmann is largely responsible, namely, block or parenchymatous injections, was welcomed by workers in the anatomic field; by merely plunging the syringe into blocks of tissue or into the parenchyma of organs many small vessels could be filled and traced. When, later, a pressure apparatus was substituted for the syringe larger fields could be injected and further topographic advances were made.

The method of Gerota, presented in 1896, created a new era in the development of lymphatic investigation. Gerota recommended that the tissues be injected through a fine glass canula under pressure with a mixture consisting of Berlin blue, turpentine and ether. This technic, endorsed by Poirer, Most and other authorities, permits the tracing of the injected matter over greatly enlarged fields down to the smallest capil-

laries, and the collecting trunks can be traced to the receiving lymph nodes.

It is only since the work of Von Recklinghausen, who discovered that the lymph spaces and lymph vessels were lined with endothelial cells, that we possess a somewhat modern conception of their structure. Their embryologic origin has been in doubt until almost the last decade. Sabin has determined that the first lymphatic elements to appear in the developing ovum are budding processes from the endothelial lining of the veins, which by innumerable branching processes invade the body tissues down to the ultimate capillaries. It has been conclusively shown that the progress of the advancing capillary is by ameboid cellular outgrowths of the growing endothelial cells (E. R. Clark), and that actual growth is accomplished by cell division in the endothelial wall. The first endothelial bud sprouts out from the anterior cardinal vein to form the anterior lymph sac. An increasing series of buds unite to form plexuses which develop into additional sacs—"lymph hearts"—and from these organs continually advancing capillaries spread into the the developing fetal tissues.

Accounting for the peculiar characteristic anastomosis of lymph elements, it is interesting to note that investigators observing these advancing vessels under the microscope have demonstrated that the growing lymphatic tip has the remarkable chemicophysical power of avoiding blood capillaries while having an attractive affinity for lymphatic vessels.

The lymph glands have two sources of origin, according to embryologists: (1) A primary group formed from the embryonal lymph sacs by the bridging of numerous bands of connective tissue covered with endothelial cells. These nodes are found in chains lying along the course of the larger and earlier formed blood vessels. (2) A secondary group developing along the course of the larger lymph vessels by the formation of plexuses into sinuses and the subsequent bridging of the sinuses with lymph cords—follicles forming in the resulting trabeculæ. These glands are found in the developed body with no apparent relation to preformed venous structures.

III.

The difficulties encountered in the injection of the smaller lymph vessels of the trachea are several in number. The mucous membrane is firmly adherent to the subjacent cartilaginous rings which are imbedded in a strong dense fibroelastic membrane, and in comparison to the mucous lining of the nose is remarkably thin. Great difficulty has been experienced in attempting the injections in consequence of this firm, tense attachment. With injections carried out on the cadaver the principal objection has been the rapid destruction of the thin mucous surface due to evaporation and autodigestion. In the experimental injections made in living animals, after exposure of the trachea by open operation, results have been unsatisfactory because of the escape of the injected fluid into the tissues and because the operative procedure introduces the foreign substance into artificially opened lymph spaces and vessels, producing innumerable artifacts and errors which render subsequent deductions questionable.

It has occurred to me recently that probably many of the difficulties might be overcome by endoscopal injections of a proper substance into the tracheal mucous membrane of living animals with examination of the resulting absorption at varying periods postmortem. This method would obviate the necessity of injuring the windpipe and peritracheal tissues and, without interfering with the normal physiologic conditions and functions of the organ, would permit investigation of the real network of origin of the lymphatics in its mucous membrane.

Dogs were chosen for most of the present experiments, on account of the more favorable diameter of the lumen of the trachea, rabbits proving too small for satisfactory endoscopic work. Young dogs were found in preliminary investigations to lend themselves more readily to the work than adults, it being noticed in many instances that the mucous membrane was less firmly attached to the inner surfaces of the cartilaginous rings than in older animals. It was also remarked that though the normally thin membrane was if anything more thin in younger tracheas, they possessed, on the other hand, the valuable asset of greater elasticity, the membrane of adult animals

proving very friable with small submucous injections. Thin dogs were considered to be the most desirable, as it was possible to control the position and movements of the trachea externally with the hand, and short necked animals were found to allow more exacting control during the actual injecting, as they permitted shorter shanked needles to be used with consequent better leverage.

The following individual methods of technic were devised and carried out with varying degrees of success:

(1) The animals were anesthetized with ether after the administration hypodermatically of morphin, and the Brüning electroscope was passed into the trachea. Injections were made with a Record syringe fitted with suitable sizes of the long shanked needles used at one time by Dr. Lynch and possibly still used by him for injections into the larynx. It was found to be essential that the needle point be rather short and of fine caliber in order to prevent laceration of the mucous membrane, and this danger was further guarded against by having the needle point bent into its shank at an angle of about fifteen degrees, thus allowing introduction of the point approximately parallel with the surface of the membrane. As a precaution against too great a pressure of fluid from the syringe the plunger was discarded and a small rubber bulb was capped over the open end. In this way the injection was more readily controlled, the rate of flow being easily regulated by gentle pressure of the fingers on the bulb.

The first injected substance used consisted of a thoroughly agitated 25 per cent solution of fresh India ink. It seemed advisable to dilute the solution, the stock ink being apparently too thick and oily to pass readily through the minute lymph vessels. Small quantities of the suspension varying from one-tenth to one-quarter of a minim were used after the first injection, larger amounts rupturing the mucous membrane with a spattering of the black fluid over the surface of the trachea. The ink injections proved to be of only partial value, as was to be expected. The lymphatic network could only be determined in small areas immediately surrounding the point of injection and only after almost immediate autopsy. In autopsies made eighteen to twenty-four hours after injec-

tion the ink was found only at the point of needle entry. In some of these cases, however, we were able to demonstrate large collecting vessels outside of the trachea by reason of engorgment with carbon bearing lymphocytes; and in all the cases carbon deposits were found by microscopic examination in the glands of the recurrent chain and deep internal jugular nodes.

(2) The same technic was carried out and a far more satisfactory demonstration of the lymphatic network was obtained by substituting for the India ink an aqueous solution of Berlin blue. This stain, which has a special predilection for endothelial cells was made according to the following formula:

Berlin blue	1 part
Oxalic acid	1 part
Water, distilled	12 parts

In autopsies made within two hours after injection the lymph vessels show up as a fine blue radiating network, the details of which are visible to the naked eye and very clearly defined with an ordinary reading lens.

(3) The experiment was repeated using a one-half of 1 per cent solution of silver nitrate after the method of Golgi. The method was discarded after a few trials, as the medium permeated the tissues indiscriminately, and after exposure to sunlight stained the mucous membrane more like a contrast stain than a selective one.

(4) Intratracheal injections of India ink were made through a needle plunged into the trachea between two of its upper rings in a manner similar to the method used by Blake and Cecil for the introduction of pneumococci into the trachea. In these cases no discoloring of the lymphatic vessels could be determined at autopsy three hours later, but it was evident that some lymphatic absorption had occurred, as carbon particles were found on microscopic examination of the peritracheal lymph nodes. Animals treated in this way were kept for a time under close postoperative observation, and in nearly all instances black streaks were apparent in the mucous secretion from the mouth after the first half hour. It is therefore probable that a very large factor in the disappearance of inert

substances injected into the lumen of the trachea is not absorption but the normal physiologic action of the ciliated epithelium of the tracheal mucous membrane.

(5) This technic was repeated, substituting Berlin blue for India ink. There was no macroscopic evidence of absorption and none could be determined under the microscope.

(6) In an effort made to secure a larger field of injection the endotracheal method described above (2) was repeated with modifications. After introduction of the electroscope a fine dry needle was inserted externally through the tissues of the neck into the tracheal lumen. After localizing the position of the point with the eye the needle was withdrawn from the lumen and, guided endoscopically, was reinserted into the lateral wall. This maneuver permitted the use of a longer needle point, the axis being parallel with the interspace, and there was consequently less danger of back flow along the course of the needle track.

(7) In conjunction with the latter method, more through accident than by design, a large quantity of the blue stain was injected into the peritracheal tissues, and postmortem examination was made in one case after one hour, and in a second at twenty-four hours. In the first instance there was a general flooding and staining of all the membranous structures, the dye having spread upward to a short distance from the occipital bone and downward to the bifurcation—guided by the fascial planes. The second case (after twenty-four hours) showed a blue tinted discoloration at the needle puncture, but the only evidence of flooding of the peritracheal spaces was a faintly green tinge in the membranes. The probable explanation of this phenomenon is that the stain, passing through the lymph spaces, entered the lymphatic vessels and, attacking the nuclei of the endothelium, caused a subsequent desquamation, the liberated cells carrying off the foreign matter.

In general the injections were made in groups of three at two levels; three in the upper trachea within two centimeters of the cricoid ring, and three just above the bifurcation. Each set consisted of one in the anterior wall, one in the lateral wall, and one posterior injection, the latter being made in the membranous strip between the open portion of the tracheal rings.

It must be stated in fairness to the reader that only a small proportion of the many injections made proved of value. In some instances no traces of the supposedly injected fluid could be found, due probably to an overzealous attempt to inject very minute quantities to avoid rupture of the vessels; and many times it was found postmortem (especially before a short needle was used) that the point had penetrated the tracheal wall and emptied its contents into the loose areolar tissue of the peritracheal spaces. In many cases the needle, however gently manipulated, tore through the delicate mucous membrane and the stain was spattered freely into the lumen of the trachea. Even in many of the successful experiments the limitations of the various methods used were apparent. The lymphatic course could be followed in most instances for only a relatively short distance from any one point of injection, as far as the superficial network was concerned, though it was possible to illustrate the continuous routes of absorption in many of the experiments, connecting the tracheal efferents with their receiving nodes in the general lymphatic system.

IV.

A study of the findings in a large number of tracheal injections indicates:

(1) That there are practically no lymph vessels other than the fine intercellular lymph spaces in the mucous membrane where it passes over and is attached to the cartilaginous rings.

(2) The tendency of lymph flow in this network of origin on the surface of the cartilages is upward and downward to the nearest interspaces.

(3) When the injected fluid reaches the interspaces (or, as most frequently occurred, was injected directly into the interspaces), the course of absorption becomes horizontal, running into small vessels lying between the rings. These tiny trunks vary from one to three in number, usually single in the cervical portion and increasing at lower levels of the tube.

(4) The lower ring surfaces are somewhat more easily injected and the fluid shows a tendency to spread with equal ease to the next upper or lower interspace. Higher in the trachea there is a distinct tendency for the absorbed pigment

to migrate upward to the first neighboring interspace rather than downward. It is also noted at the central portions of the tube that the superficial network is consistently less dense than at either the upper or lower levels.

(5) In the anterior wall injections the fluid crosses the midline with ease and shows an equal tendency to spread horizontally to both lateral walls, with a marked inclination to circumscribe the tube.

(6) The lateral injections, and this holds for both upper and lower levels, may spread anteriorly, but the characteristic tendency is posteriorly to reach the membranous portion of the trachea.

(7) The posterior injections into the membraneous wall demonstrate the presence of a large number of lymph vessels, and larger individual vessels at any given level compared to the number and size of the vascular elements in the cartilaginous portion of the tube. Here also is apparently a greater tendency to upward and downward communication, the incomplete cartilages offering no obstruction here. In the pars membranacea, it may also be demonstrated, the lymphatic vessels are more numerous and of larger caliber in the thoracic portion compared to higher levels.

(8) The horizontal collecting vessels are more numerous in the lower interspaces and seem to increase in caliber in the interspaces near the bifurcation. They are joined in their course by minute collectors from the surface of the two adjacent rings, and the general direction of their flow is toward the posterior membranous wall. There is a disappearance of the majority of these trunks beyond the open ends of the cartilaginous rings in a radiating network of fine vessels which cover a comparatively large area in the membranous wall—a network which usually extends across the midline to similar vessels on the opposite side. It is here, according to Poirer, Most, and a few other investigators, that the efferents leave the tracheal wall to join the general lymphatic system.

(9) Though there is a vast anastomosis of these vessels in the peritracheal connective tissue between the windpipe and the esophagus, by far the greater portion of the main trunks join the lymph vessels accompanying the recurrent laryngeal

nerve. These large collectors are interrupted in their course by nodes—the paratracheal glands—corresponding to the recurrent chain in man. From these nodes drainage to the deeper internal jugular groups can be traced by the deposits of the carbon particles of the India ink.

CONCLUSIONS.

There is a partial lymphatic absorption of inert substances by the tracheal mucous membrane in a normal uninjured and uninflamed state.

The cartilage rings, dividing the superficial network of origin into segments, offer successive lines of defense against lymphatic spread of tracheal infection.

Absorption from the mucous membrane when no inflammatory condition exists is less active in the middle third of the trachea than in either the subcricoid area or that portion approaching the bifurcation.

The tendency of lymph drainage at any given segment is to the posterior wall of the trachea, through which offending matter is carried from the field to neighboring lymph nodes.

Though the lymphatic function is segmented by the tracheal rings, and in this way may be considered regional, nevertheless anastomosis between the various levels is free by way of the plexuses in the posterior wall of the tube. There is also, to a very minor degree, an intersegmental drainage by way of individual capillaries which at irregular intervals hurdle the belly of the cartilages from interspace to interspace.

Hypothetically substituting the pneumococci for carbon particles and stains, pneumonia may be caused by absorption of the organisms through the mucous membrane of the trachea into the recurrent nodes and jugular chain, arriving at the lungs by way of the lymphatic ducts, great veins, right heart and pulmonary artery.

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Fig. 1.

Trachea of young dog; endoscopal submucous injection of India ink. Autopsy in one hour. Arrow points to site of injection. Note the enlarged black capillaries in the interspaces.

Fig. 2.

Trachea of young dog; endoscopal submucous injection of India ink. Autopsy in two hours. Magnification: 6 diam. (X marks anterior wall.) Shows tendency of the pigment to be carried to the interspaces and toward the membranous part of the tube. The specimen is is taken from the subcricoid area.

Fig. 3.

Trachea of dog; submucous injection of India ink. Autopsy in one hour. Magnification: 6 diam. Notice here the marked tendency of the pigment to pass through lymph vessels in the interspaces and toward "X," which marks the direction of the posterior wall. The specimen is taken from the suprabifurcation area.

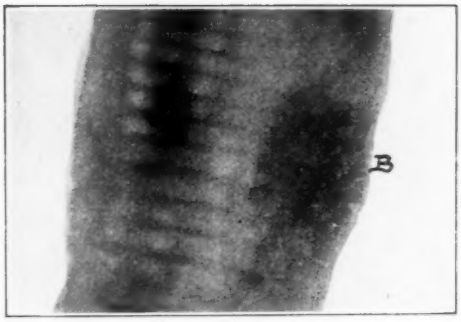

Fig. 4.

Trachea of young dog; submucous injection of Berlin Blue. Autopsy in one hour. Actual size. "A" marks the point of injection and at "B" is seen the stained network of the membranous portion of the wall through which the horizontal collecting vessels become extratracheal.

Fig. 5.

Trachea of young dog; submucous injection of India ink. Autopsy in three hours. "B" marks the point of injection. "A" indicates the lymphatic cords accompanying the recurrent laryngeal nerve—engorged with carbon bearing lymphocytes.

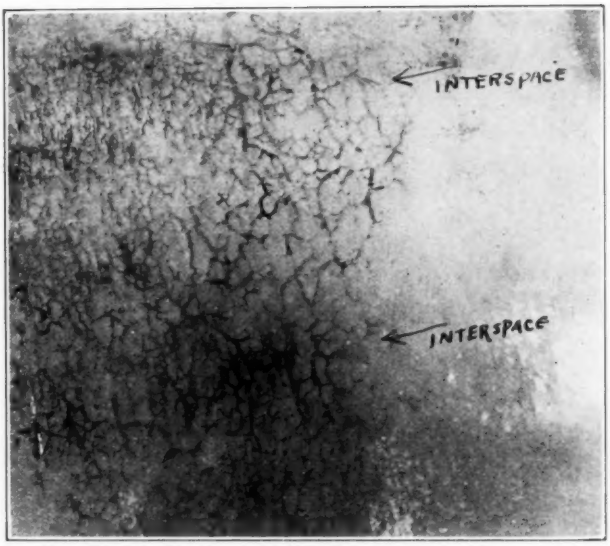

Fig. 6.

Trachea of young dog; submucous injection of Berlin Blue. Autopsy in 30 minutes. Magnification: 16 diam. Showing the rich dense network of lymphatics near the bifurcation. Here also is seen the tendency to absorption through the interspaces, the general direction being from the point of injection to the posterior wall.

XLIV.

MALIGNANT TUMOR OF THE TEMPORAL BONE.

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In the period of twenty years of practice limited to the diseases of the ear, nose and throat, I have had opportunity to observe and operate upon three cases of malignant tumor of the ear. The first case, sarcoma, I described in 1908 in the American Archives of Otology. The second case, cancer of the temporal bone, I treated from nearly the very beginning of the disease. This case was published in 1912 in the Monatschrift für Ohrenheilkunde.

Two years ago, when in the military service, I performed a radical mastoid operation upon a Polish soldier, with chronic left side otitis media complicated by mastoiditis. Four weeks after the operation the facial palsy, exuberant granulations in the wound and the general condition of the patient became so suspicious that a microscopic investigation of the granulations was made. This investigation proved that we had to deal with a case of malignant tumor.

In all three cases mentioned above a thorough macro and microscopic postmortem examination was performed.

It is well known that the malignant tumors of the ear are exceedingly rare. Even a very busy practitioner may have no opportunity in the years of his practice to observe a single case. In the large ear clinics these cases come to view sometimes, but their ratio to the other ear diseases is very small. Bezold in his well known records of 20,000 ear patients treated in the space of 24 years had only four carcinoma cases. Sarcoma of the ear occurs still more rarely. In my article cited above, I have found in the accessible literature only three cases (two described by W. Milligan and one by Oppikofer), published from 1896 till 1908. All cases of the sarcoma of all parts of the ear (auricle, external, middle and internal ear), published till 1896, are mentioned in the work of Asch,[1] and the sum of them is less than 100.

The difficulty of picking out from literature all cases of some rare form of disease like the malignant tumors of the ear is evident from the article of Dr. O. Benesi,[2] published recently in the Monatschrift für Ohrenheilkunde, 1920, No. 11. The author affirms that the metastatic carcinoma of the ear has not been described yet ("Krebsmetastasen am Ohre sind in der Literatur nicht beschriben," l. c. page 961). Meanwhile we find in the very interesting work of Dr. Schlichter[3] four articles (Habermann, Alt, Neuman, Schwabach) mentioned, which deal with metastatic cancer of the ear!

Historia morbi of my case is very short and simple. In June, 1919, a soldier, 22 years of age, small but well nourished, was admitted to the ear department of Ujazdow Military Hospital in Warsaw. Internal organs (heart, lungs, kidneys, a. s. o.) normal. Since childhood purulent discharge from the left ear. The right ear, nose and throat normal.

The examination of the left ear revealed: Profuse purulent discharge, drum membrane absent, the drum cavity filled with granulations, red in color, slightly bleeding on touch.

The hearing power diminished—whisper, 2 meters, Rinne negative, Weber lateralized to the left ear. Caloric reaction positive. A common treatment was applied, and the patient was informed that he would be rejected on account of his ear trouble.

Three weeks later the patient's temperature rose (37.6° C.) and he complained of some pain in the ear and in the mastoid region. The mastoid process was slightly tender on pressure, the discharge became profuse and offensive in odor. This condition lasted some days and the patient was offered an operation.

On June 28, 1919, the radical mastoid operation was performed. In the mastoid process I found a small quantity of pus and granulations. The antrum was well developed and also filled with pus and granulations, as well as the tympanic cavity, which were thoroughly curetted. The ossicles (mallet and incus) were absent. The stapes was imbedded in the granulations, which were carefully removed. The cranial fossæ were not opened. Plastic after Panse method.

The course of the usual after-treatment was very unusual. The wound behind the ear was filled with a large quantity of pus and had to be reopened. The granulations were pale and exuberant; the general condition of the patient became worse. A month after the operation a sudden paresis of the left facial nerve appeared and that made me suspect that we had to deal here with a malignant tumor. The evening temperature, which immediately after the operation became normal, rose again to 38.0° C. With the double curette some particles of granulating tissue in the wound were removed and a microscopic examination made. In all preparations the same picture appeared.

Among the stroma built from the connective tissue infiltrated here and there with leucocytes we find thrown epithelial herds. In some places we can observe the junction between these herds and the covering epithelium.

The herds are of different shapes, round, elongated or branching irregularly. The cells constituting these herds show numerous abnormalities, differing from the normal epithelial cells. They are of various sizes and various shapes. The size of their nuclei is different—some nuclei are stained stronger than others. Besides this the cells show many degenerations as vacuolization and cornification, which is most distinct in the middle of the herds. (Fig. 5.)

According to this microscopic investigation, we diagnosed a cancroid, whose starting point was probably an epidermis of the external ear channel.

A similar result was obtained from a microscopic postmortem examination.

Taught by the two cases observed earlier, I called the relatives of the patient to let them know that the condition of the poor man was hopeles. The patient refused a second operation. Meanwhile the X-ray treatment had been applied (ten applications every second day—there was no radium in the hospital at that time). The treatment was without effect. On the contrary, I had the impression that the growth of the tumor was even more rapid. Two months after the operation the condition of the patient was as follows: (Fig. 1.) Total

paralysis of all branches of the left facial nerve. The left ear absolutely deaf. Caloric reaction negative. The tumor behind the ear had begun to disintegrate. The tympanic glands in the mastoid region and on the neck were swollen and enlarged—in short, it was a typical picture of a malignant tumor of the temporal bone, which I had had the sad experience to observe already twice before.

Three and one-half months after the operation the wound looked like a large, deep hole, with the walls covered with pale abundant masses of tumor and pus. (Fig. 2.) As we see on this figure the auricle was held in situ by a relatively thin piece of healthy skin. Some weeks later this piece was destroyed by the disintegrated tumor masses and the auricle fell off at the change of dressing. (Fig. 3.) Constant headache appeared and grew more severe every day—a sign that the tumor had involved the dura. Morphin was administered and it was necessary to make many injections day and night. General cachexia which could be noticed already on the second month after the operation developed and was the cause of the exitus on December 18. The patient was in the hospital about six months.

Postmortem examination showed following changes:

Exhausted, emaciated body of a young man. No pathologic changes in the internal organs; no metastases. In the region of the left ear a great, deep wound involving a space between the zygoma and occipital bone. Practically the whole temporal bone, except a part of the pyramid, was destroyed and in place of it we found masses of tumor. Left auricle was absent. After removal of the roof of the skull and of the brain it was evident that the external surface of the dura in the region of the temporal bone was covered with tumor masses which did not penetrate through the dura into the cranial cavity. The brain was anemic, but without any changes. After removal of the dura it was seen that the tumor masses had destroyed not only the remnants of the mastoid process (after the radical operation) and the middle ear, but also the part of the pyramid. The part of the pyramid which remained (Fig. 4) had the appearance of a small process, covered with a thin layer of the tumor mass penetrating the bone.

The lymphatic glands on the neck were enlarged, but only in the immediate neighborhood of the destroyed temporal bone. The lymphatic glands on the other side were normal.

As we see from the postmortem examination, in this case we had to deal with the malignant tumor limited to the left temporal bone without any metastases, involvement of the brain, as was described in other similar cases. The only cause of the exitus was cachexia, as in my case of the carcinoma cited above. In the case described by Danziger,[4] the tumor masses, after destroying the left temporal bone, penetrated through the dura into the cerebrum, where in the left temporal lobe they formed a large growth. This growth caused aphasia and was the immediate cause of the exitus. In my first case of sarcoma, cited above, the tumor masses destroyed the processus odontoideus of the second vertebra, which was broken when the patient sat up in bed. The processus odontoideus entered into the cranial cavity and injured the medulla oblongata, causing the sudden death of the patient. Usually, as we see from the literature, the common cause of the exitus in these cases is a cachexia.

How should we operate a case of a malignant tumor of the temporal bone, if we operate at all? In all three cases upon which I operated, a radical mastoid operation was made, which proved unsuccessful and obviously insufficient. As we see in the literature on the subject, there is only one case, that of Jansen,[5] in which eight months after the radical operation no relapse had appeared. In the article of Grasser[6] a very interesting case is described so far as that a diagnosis was made by paracentesis of a reddened and bulging drum membrane. Through the opening in the drum, masses of malignant growth protruded. In this case, after operation there was no relapse in the space of eight years. About the malignant tumors of the ear Doyen in his "Traité thérapeutique chirurgicale," Vol. II, writes that it is impossible to operate upon such cases: "Ces néoplasmes sont au dessus des ressources de chirurgie"; "l'opération radicale est impossible dans l'état actuel de la chirurgie."

I think that an early operation, consisting of total resection of the whole temporal bone, including the pyramid, may be

successful. In the case described above, nature shows us the way to follow when operating. Despite such an immense destruction of the part of the skull, during all the time not even the slightest bleeding occurred, although the tumor laid open all the big blood vessels (carotid artery, sigmoid and petrosal sinuses, bulb of the jugular and jugular vein). The early facial paralysis, the most prominent symptom of the malignant tumor of the ear, teaches us that when operating we must sacrifice this nerve. The involvement of the pyramid shows that we must resect the whole pyramid up to the internal ear channel.

Such an extensive operation is not only technically feasible, but, as I know, has been performed three times, once with successful result.[7] The indications for these operations were twice a destruction of the pyramid by a bullet; once the operation was performed accidentally on account of necrosis of the pyramid (Ruttin, cited in the article of H. Frey.[8]) In the interesting article of H. Frey, the author describes briefly the method of the total resection of the pyramid and says that such an operation can be indicated above all in the cases of malignant tumors of the temporal bone. The technic proposed by H. Frey consists in short in a radical mastoid operation, after which the bony attachment of the pyramis should be cut away and then the pyramid removed in toto.

My experience, limited to the performing of this operation on many specimens, has taught me that the most difficult step in this operation is the laying free of the dura, which must unavoidably be injured when removing such a large block of bone as a pyramid. We can work without injuring the dura only in one manner, namely, by chiseling carefully and removing every splint of the bone after a previous thorough separation of the dura (Fig. 6).

I should propose in a case of early recognized malignant tumor of the temporal bone such an operative measure. (The particulars I will describe in a special article.)

A. Preliminary operation.

1. Thorough radical mastoid operation. The dura of both cranial fossæ must be laid free as far as possible.

2. Ligation of the carotid artery and of the jugular vein. Removal of lymphatic glands.

B. Some days later the resection of the temporal bone.

1. Blocking of the sinus by using the method of Whiting-Meyer.

2. Laying free the facial nerve, which will serve us as a guide to the internal ear channel.

3. Removal of the posterior part of the pyramid until the vestibulum and internal ear channel have been reached.

4. Removal of the upper parts of the pyramid. Cave sinus petrosus sup.

5. Removal of the cochlea and of the inferior and anterior parts of the long external auditory canal and of the drum cavity. The opening of the eustachian tube is laid free.

6. Resection of the facial nerve and removing of the bone in which the facial canal is located.

7. Total resection of the auricle with the tragus and cartilaginous portion of the external ear channel.

On the figure 6 we see the specimen with the operation made.

I believe that such an extensive operation, when performed early, can be successful, as the total resection of the larynx in the cases of laryngeal malignant tumors. The facial paresis can be corrected by means of an anastomosis with the hypoglossal nerve, which should be made some weeks later.

When we bear in mind that the prognosis in cases of malignant tumors of the temporal bone is absolutely unfavorable, it is clear that only such an extensive operation, with removal of all parts in which the tumor can grow, may be employed when we intend to operate upon such cases at all. Every other operative measure will always prove insufficient and cause only the more rapid course of the disease.

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Fig. 1.

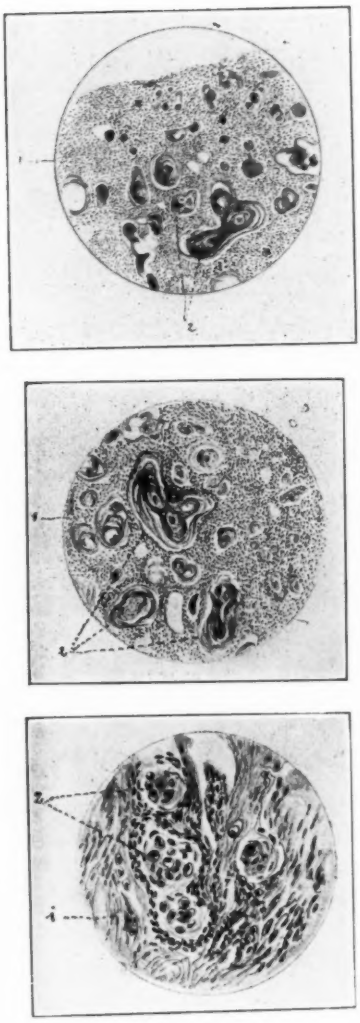

Fig. 2.

Fig. 3.

Fig. 4.

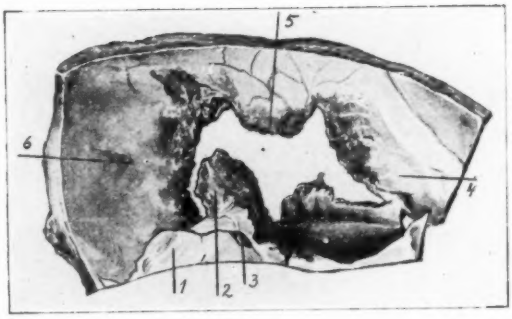

Fig. 5.

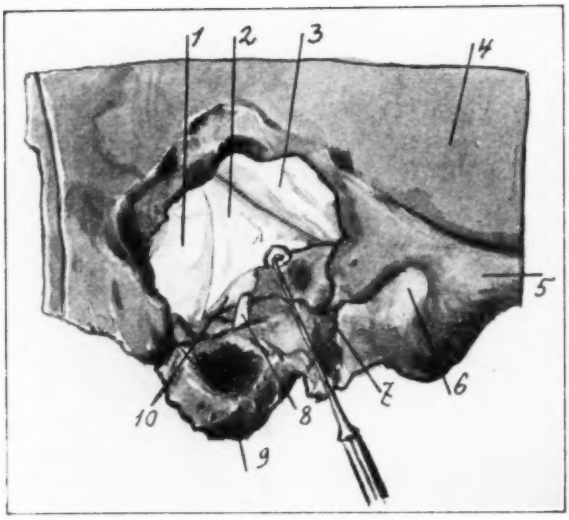

Fig. 6.

XLV.

A BRIEF REVIEW OF THE ANATOMY AND SURGERY OF THE TEMPORAL BONE, WITH REFERENCE TO THE MASTOID IN HEALTH AND DISEASE.

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I am prompted to select the mastoid for the subject of study because of its interesting development into the temporal bone; its susceptibility to infection; its unsuspected etiology in some of the more serious intracranial complications and its closely allied clinical manifestations of other diseases which frequently are deceptive to the physician.

All students of medicine, as well as the surgeon, frankly acknowledge that it is most difficult to comprehend and to clearly visualize the anatomy of the temporal bone and the vital structure within and adjacent to the mastoid, during the process of dissection or operation.

In view of these facts and the urgent necessity for a thorough familiarity with the delicate anatomic structure of the mastoid, liable to irreparable damage during operation, I shall present photographs of original dissections of the temporal bone and of the mastoid, descriptive of their beginning in the fetal stage, trace their early development in the quite young and continue until it becomes an integral of the temporal bone in the matured adult.

In utere, the periotic capsule is cartilaginous. Ossifying material is not deposited until the end of the sixth month. This is done in four centers, according to their relation to the capsule. The epiotic is the last to appear, and is first seen at the posterior part of the posterior semicircular canal; and from this center the mastoid process takes its rise

and that is the only center to be given consideration in this paper.

In the new-born there is a complete absence of the mastoid as is seen in Figures 1, 2, 3 and 4. The first suggestion of such a process presents itself as a small tubercle, behind the upper part of the pars tympanica. That part of the petrous bone from which the mastoid process develops presents a surface so flat as merely to suggest a prominence. The development of the cell space in the mastoid proceeds by cellular proliferation. Beginning at the posterior superior periphery of the antrum, it advances towards the transverse sinus.

The mastoid antrum is the only pneumatic space present in the temporal bone in the new born and is larger than that of the adult. The mastoid antrum opens into the tympanic attic, a small chamber lying in the upper and back part of the tympanum. Its size varies with age; it is present in birth and increases until about the third or fourth year when it gradually diminishes, owing to the encroachment of the bone developed around it.

The squamo mastoid suture lies between the anterior surface of the tuberculum mastoidum and the posterior portion of the pars squamosa and forms the external wall of the mastoid antrum in the new born as shown in Figure 4. It gradually disappears during the first years of life, although it may persist in the adult as a jagged, deep furrow on the external surface of the mastoid process. In such case it may be described as representing the appearance of a depressed fracture.

The roof of these chambers is formed in part from the squamous and in part from the petrous shown in Figure 2 (a) and (c). The suture passes directly through the tegmen tympani and antri on the inner surface at the junction of the petrous and the squamous, Figure 2 (a) and (c). It is quite patulent and for this reason susceptible to meningeal symptoms in case of acute suppuration of the middle ear.

The outer surface of the temporal bone in the new born lacks the osseous external meatus seen in the adult. The membranous meatus is connected with the shallow rim of bone, Figure 1 (b). The pars tympanica to which the membrana tympani is attached is shown in the groove, Figure 2 (b). It is the close relationship between these membrances that makes a child sensitive to pain upon the manipulation of the auricle during an attack of acute otitis media. Owing to the patulent condition of the pars squamosa and pars petrosa, Figure 4 (a) and (c), the escaping pus in an acute suppurative otitis media causes postauricular swelling so frequently seen in the very young.

The outer wall of the antrum is formed by a plate descending from the squamous, Figures 1 (a) and 2 (a). This wall is very thin in early life, but is developed by deposits from the periosteum. The depth of the antrum increases from the surface.

The roof of the antrum, tegmen antri, is merely the backward continuation of the tympanum. The level of the roof is indicated by the posterior root of the zygoma. The level of the floor of the adult skull at the tegmen antri is, on an average, less than one-fourth of an inch above the external osseous meatus.

In the development of the temporal bone after birth, the two conspicuous changes brought about are the formation of a mastoid process and of a bony external meatus, or rather the development of the tympanic plate of the temporal bone. During the first year of life this tubercle takes a downward course. This is caused partly by its own growth and partly by the action of the muscles. The mastoid process does not assume its adult form until the third year of life (Figure 5).

In some cases the mastoid is massively developed; in others, reduced to a short protuberance. The cells may be of the pneumatic or of the diploetic type, or they may be mixed in form. In fact, innumerable varieties exist. The pneumatic cells of the mastoid process are lined with a delicate, gland-

less membrane, a continuation of the mucous membrane of the tympanic cavity.

The antrum communicates with the lower vertical cells of the mastoid through its floor. The floor being on a lower level than the opening into the tympanum, drainage of the antrum is difficult and the fluid readily filters into the lower cells.

The bend of the sigmoid part of the lateral sinus (Figures 8 b, 8c and 29) with its descending portion is seen behind the mastoid antrum and cells. A vertical line drawn one-fourth of an inch behind the posterior bony wall of the external auditory meatus and between the levels of the floor and the roof will indicate, in the majority of cases the position of the anterior convexity of the sigmoid sinus in the adult.

If the mastoid cells be of the pneumatic type (Figures 26 and 32), the lateral sinus will usually be found deeply situated and more posterior, hence less liable to exposure or injury; whereas, in the diploetic type (Figures 8a and 29) of cells, the location of the sinus is invariably reverse. This has proved characteristic in chronic purulent inflammation as revealed in the radical mastoid operation. It is not uncommon to find that the posterior osseous wall of the auditory canal forms the anterior wall of the sigmoid portion of the sinus and is frequently exposed or injured at that point. The lower and vertical cells of the mastoid are developed later than those of the antrum and vary in their contents. Only about 20 per cent contain air. The foramen, an opening for the emissary vein (Figures 5a) is on the posterior border of the mastoid process.

I consider roentgenography one of the most valuable single aids in the study of the mastoid in health and disease. Experience and great skill are required to interpret the shadows of the picture; but that test ought to be a part in the study of every case in determining whether or not to open the mastoid. Any abnormality in the development of the mastoid; the type and extent of the cell area; the character of the osseous structure; the position of the lateral sinus; or any chronic or acute necrotic erosion of the mastoid and its adjacent structure will be revealed by the roentgenogram.

The accompanying radiograms well serve to illustrate those different conditions. Figure 25 is that of a physician who had an acute purulent mastoiditis with necrotic erosion revealing an extradural abscess. Undoubtedly, the cells extending over the zygoma were of the type shown by Figures 8, 8b, 13, 20, 23 and 24.

Humanity owes to the scientific medical profession an everlasting gratitude for the vaccines and serum therapy. Diphtheria and typhoid fever are no longer numbered among the chief predisposing causes of middle ear infection with mastoid complications. Statistics prove measles, scarlet fever, pneumonia and influenza fruitful sources of middle ear infection. Let us hope a serum to prevent or annul the virulence of those diseases will soon be discovered.

Great precaution should be taken to protect the ears when swimming and diving, lest water enter the middle ear by the way of the eustachian tube or by a patulent ear drum and superinduce mastoid involvement or intracranial complication. This is especially likely if the ear drum be patulous from an existing otorrhea or if cholesteatoma be present. Infection of the middle ear demands prompt surgery. Often a minor operation in the early stage of inflammation will prevent serious complications and a major operation. Any inflammatory process of the middle ear becomes at once a surgical case.

Reviewing my clinical experiences, I recall with surprise the frequency of mastoid infection following tonsillectomy and submucous resections, and the utter indifference patients manifested concerning diseases of the ear. A very great majority of patients coming to our clinics may be grouped in four classes:

1. Those suffering acute pain.

2. Those with acute otorrhea, entirely oblivious of the thought of complications. More than 50 per cent of this class cannot give an intelligent history of the onset of the disease; its cause; its duration or its treatment.

3. Those who are rapidly becoming deaf; others who have vertigo, and those threatened with or perhaps have developed intracranial complications.

4. Those who have lost the function of hearing and are

being outstripped in their vocations by others not thus handicapped.

In a series of two hundred and fifty mastoid operations, there seemed to be no age limit; I have operated on a three months' old infant and on a patient seventy-two years of age.

The right or the left mastoid was involved about an equal number of times; also, neither the acute nor the chronic type of mastoid prevailed, the number of operations in both cases being about equal. That in the acute form of the disease, the streptococcus and the pneumococcus were the predominating type of infection; in the chronic cases there was a mixed variety. In one case of chronic mastoiditis complicated with septic sinus thrombosis the colon bacillus was found.

Operations were performed in a few cases complicated with pneumonia, tuberculosis, the exanthematous diseases and diabetes. In the diabetic case, 10 per cent sugar was found in the urine when admitted to the hospital. As this was an emergency, operation was done under local anaesthesia, using Schlech's fluid; in another case a radical mastoid operation was done under morphine and scopolamine, socalled Twilight sleep. Gas anaesthesia was used in the tubercular case.

The facial nerve due to necrotic erosion of the fallopian canal (Figures 31 and 32) was found exposed at the time of the operation. There was no preexisting facial paralysis, nor did paralysis ensue; whereas, if the facial nerve be exposed or injured during the operation, paralysis of the face is most likely to occur.

The frequent necrotic exposure of the dura with a moderate degree of localized thickening or extradural abscess shows that nature will in some instances check the invading organisms by a protective barrier. The sigmoid sinus passing through the mastoid as it does is in great danger of infection by necrotic erosion. Here again, nature protects the vessel wall by a barrier of granulations. Septic sinus thrombosis does occur, however, and death is inevitable, unless prompt surgical relief is instituted.

It is the consensus of opinion that the best preventive of chronic otorrhea is opening the mastoid before intracranial complications ensue. I cannot recall a single case of mastoid disease, either the acute or chronic, where there were no complications and death ensued, that might not have been averted if the operation had been performed early.

Drooping of the posterior wall of the external auditory canal is regarded as pathognomic of mastoid involvement by some authorities; others equally well informed claim this is not an infallible indication for operation. This is undoubtedly true in that type of case where pus is confined in the cells of the tip or lower portion of the mastoid. In such cases operation is as much indicated as if all the classical signs were present. Be obedient, therefore, to the homely adage, "Wherever there be pus, let it out."

Textbooks enumerate pain, tenderness, redness and swelling over the mastoid as the classical symptoms of mastoid involvement. Usually those symptoms are present; but their obsence does not prove that the mastoid is not seriously involved. For instance, a mastoid with a thick sclerosed cortex never shows those well defined symptoms because the internal resistance is less than that in the external direction.

I have seen patients with a very extensive necrosis and in a general septic condition, with practically a normal temperature and with no tenderness over the mastoid even upon very hard pressure, who proved upon operation to have had a perisinus or an extradural abscess, or both. If the mastoid had not been opened intracranial complications would have resulted, without doubt.

I believe the hearing has been preserved in many cases of acute infection of the middle ear and the mastoid when the operation has been performed early. In chronic otorrhea, the hearing may or may not have been impaired as much depends upon the character of the involvement of the middle and internal ear. Loss of hearing is likely to follow operation because much of the structures employed in the conduction of sound waves has been removed. But in my experience the end results have been gratifying.

ILLUSTRATIONS.

FIGURES 1, 2, 3 AND 4

represent dissections of the temporal bone in the fetal stage.

Figure 1 presents the three portions of the fetal temporal bone:

(a) The pars squamosa which is the thin porous shell-like bone, the lower portion forming the outer wall of the epitympanum.

(b) The pars petrosa which is detached from the pars squamosa and pars tympanica. It presents the inner wall of the tympanum, the upper half representing the recessus epitympanicum and the antrium.

The lower half presents the recessus mesotympanicum. The promontory is readily seen. The fenestra vestibula with the stapes inserted is in the superior posterior portion, and a few millimeters below, is seen the fenestra cochleae which opens into the first turn of the cochlea. It is closed with a membrane called the membrana secunda and it separates the tympanum from the scala tympani.

FIGURE 2—A, B AND C.

Figure 2. This is the inner surface of (a) the pars squamosa with (b) the pars tympanica attached, which represents an incomplete ring with a groove for the insertion of the membrana tympani.

The malleus and the incus are held in position by part of their ligamentous attachments. The head and neck of the malleus and the body of the incus repose in the recessus epitympanicus. The stapes is detached from the process of the incus and is located in the fenestra vestibuli as described in Figure 1 (c).

The outer wall of the recessus epitympanicus ad antri is formed by the articulating surfaces of the pars squamosa and the pars petrosa.

FIGURE 3

Presents the articulation of (b) pars tympanicus to (C1) pars petrosa.

The malleus, the incus and the stapes are held in position by their ligamentous attachments. The fenestra rotunda is seen on the inner central border of the pars tympanica and the stylomastoid foramen for the exit of the facial nerve is on the outer posterior border, while in front and below, is shown the foramen for the carotid artery.

C2 presents an unobstructed view of the fenestra vestibula and the fenestra cochlea; the promontory; the tympanic end of the eustachian canal and the groove immediately above to which is attached the tensor tympanic muscle.

Above the fenestra vestibuli is seen the osseus capsule of the horizontal semicircular canal; above and posterior to this, is the antrum.

FIGURE 4

Shows three sections, (a) the pars squamosa; (b) the pars tympanica; (c) the pars petrosa of the fetal temporal bone have united. The Rivinian notch (d) is clearly defined. The ossicles are held in position by their ligamentous attachments; (e) the petrosquamosal articulation is revealed by the fissure on the posterior border; (f(the stylomastoid foramen is seen on the posterior border of the pars tympanica; (g) the carotid foramen is at the anterior inferior border; (h) the eustachian foramen is at the apex; (i) the Glaserian fissure is clearly shown.

The extremely porous condition of the bone in the fetal stage is noted.

FIGURE 5—A, B, C AND D,

Represent the temporal bone with the early development of the mastoid in the child.

Figure 5 (A) shows the early development of the mastoid in a child. Below the tip is the depression of the digastric groove. Between the tip of the mastoid and the posterior border of the pars tympanica is the stylomastoid foramen for the exit of the facial nerve. The dark notch immediately below is the jugular bulb. Upon opening the mastoid, greatest care must be taken not to sever either the nerve or the jugular bulb. The notch on the posterior border of the mastoid is the foramen through which passes the mastoid emissary vein.

The pars tympani has developed to a considerable size and forms the tympanic plate in later life. The Glaserian fissure is the depression between the pars tympanica and the articulation of the condyloid joint.

Figure 5 (B). This gives a view of the pars petrosa as the bone is lying on its concave surface.

(C). The large foramen is the carotid canal.

(D). The small foramen is the stylomastoid for the exit of the facial nerve, and (E) the depression between them is the jugular bulb. This again shows how readily the nerve and the jugular bulb may be severed. The mastoid is seen as a slight elevation.

Figure 5 (C) gives a view of the posterior surface of the petrous, the concave surface up. The deep groove of the lateral sinus is clearly seen ending at the jugular bulb, just posterior to the carotid foramen and is indicated by the large dark foramen. The superior and posterior semicircular canals are opened and are represented by the two dark lines above the jugular depression. The facial nerve is represented by a dark line entering the internal osseous meatus as seen in the upper part of the petrous, directly above the jugular foramen.

Figure 5 (D). Viewing the anterior convex surface of the temporal bone. The dark foramen is the continuation of the carotid canal. Next is the lamina spiralis exposed showing the course of the facial nerve as it passes over the first turn of the cochlea to enter the fallopian canal on the inner anterior wall of the tympanum. The top foramen is the exposure of the superior semicircular canal.

The tegumen tympani and antri are opened, exposing the incus and the head of the malleus.

The dark irregular surface just above the petrosa is the transverse sinus. The long slender lines with branches are the grooves for the middle meningeal artery on the inner surface of the squamosa.

FIGURE 6.

The fully developed temporal bone and mastoid (Figure 6) with its massive zygomatic bar and root. The squamosa, the petrous with the mastoid as its base, and the tympanic plate which has developed from the incomplete ring of the infant (Figures 1b, 2b and 4b).

The Glaserian fissure is posterior to the condyloid fossa; the silkworm gut projecting from the fissure represents the corda tympani nerve.

Macewen's triangle is formed above by the posterior root of the zygoma; the anterior boundary, by the posterior auditory osseous canal. The third or posterior line of the triangle is an imaginary line joining these two lines. This triangle was considered of great importance in former years in trepanning the mastoid to drain the purulent process, as it was the most direct approach to the antrum without injury to the lateral sinus.

When removing the cortex of the mastoid, the operator must never go above the posterior root of the zygoma lest he open into the middle fossa of the skull and injure the brain.

FIGURE 7.

The temporal bone divided through the petrous. On the left side is seen the ear drum, the incus and the malleus. The tensor tympanic muscle is shown attached to the malleus.

The corda tympani nerve is represented by a fine light line crossing the membrana tympani at the end of the process of the incus.

On the right, the stapes is seen in the fenestra ovalis with the stapedius muscle inserted in the neck of the stapes.

The osseous capsule of the horizontal semicircular canal is seen above the upper border of the nerve.

The dark lines on the promontory are the tympanic branches of the glosso-pharyngeal, or Jacobson's nerve.

The heavy dark line between the horizontal canal and the fenestra ovalis represents the course of the facial nerve.

Note the mixed type of mastoid cells.

FIGURE 8—A, B AND C.

Figure 8 (A). This represents the temporal bone with the cortex removed from the mastoid, showing the mixed type of cells, the diploetic predominating. The cells extend over the zygomatic bar as far as the tempomandibular articulation. Involvement of the cells in an acute purulent process of the mastoid would result, very probably, in intracranial involvement of the middle fossa. The dark line represents the corda tympani nerve passing through the Glaserian fissure.

Figure 25 X-ray plate shows an erosion of the squamosa. In this location an extradural abscess associated with acute suppurative mastoiditis was found.

A patient suffering from epileptic seizure of the Jacksonian type, with chronic purulent process of the mastoid involving the cells extending into the squamosa, resulting in an inflammatory irritation of the dura, recovered after a radical mastoid operation.

Figure 8 (B) shows the inner surface of Figure 8 (A). Note the cells of the mastoid extending into the squamosa. This indicates the likelihood of purulent inflammation with early meningeal irritation.

The facial nerve is represented by the dark line crossing over the modiolus and in front of the semicircular canals at the point where it enters the tympanum. The deep groove of the lateral sinus is clearly shown and is far forward.

Figure 8 (C). The cells extending over the squamosa are clearly defined. We may look for early intracranial complication in case of purulent infection of the mastoid in a patient with this type of cells. The lateral sinus and the semicircular canals are clearly shown.

The small dark spots in the petrous back of the internal osseus meatus represents the foramen for the entrance of the facial, vestibular and cochlear nerves.

The head of the malleus and the incus is seen in the epitympanicum.

FIGURE 9.

This is the anterior concave view of the petrosquamosal portion of the temporal bone. The dark lines on the convex surface of the squamosa are the grooves for the middle meningeal artery. The petrosa is opened showing the spiral of the cochlea. The semicircular canals and the epitympanicum show the articulation of the malleus and the incus. Note the thinness of the bone separating those structures from the middle fossa.

FIGURE 10.

This is the petrous with the squamous removed. The articulation of the ossicles within the tympanum are clearly seen. The dark half circular lines represent the superior and posterior semicircular canals.

Here again we have the pneumatic and the diploetic form of cells.

FIGURE 11

shows sections of the petrous bone and the spirals of the cochlea.

The internal ear is a complex chamber; the bony labyrinth contains the membranous labyrinth, and the specific receptors or sensory surfaces of the internal ear.

(b) The small section on the right side of the plate is the cochlea. It is interesting to note the calific deposits around the spiral. It would be of scientific interest to know whether or not this subject was deaf.

FIGURE 12—A AND B.

Figure 12 (a). The osseous capsule of the labyrinth.

(b) The upper foramen is the fenestra ovalis; the lower, is the fenestra rotunda. The silkworm gut represents the course

of the facial nerve as it passes between the oval window and the horizontal canal.

(c) The internal osseous canal removed with the hope of showing the foramen for the passage of the facial, vestibular and cochlear nerves. The circle on the upper surface is the superior canal.

Figure 12 (b). This shows the facial nerve (7th) passing through the internal osseous auditory meatus over the first turn of the cochlea, under horizontal canal. The spirals of the cochlea are clearly defined.

(e) The three semicircular canals are clearly defined. They serve chiefly the function of equilibration. Under the semicircular canals can be seen the oval and round windows with the promontory.

FIGURE 26.

Surgery of the temporal bone consists chiefly of opening the mastoid to drain the suppurative process and to remove necrotic bone. When the infection has extended into the internal ear or labyrinth, it may be opened to prevent further extension to the meninges.

The cortex is removed and the antrum is opened. The pneumatic type of cell is well defined.

FIGURE 27

Is the simple mastoid operation.

The upper border of the mastoid is on a level with the posterior root of the zygoma. (Fig. 6.) The large cell over the digastric ridge extends into the petrosa, opening into the jugular bulb could easily occur unless the operator has a clear visualization of the anatomy.

FIGURE 28.

This shows the exceptional beveling of the outer wall of the epitympanicum and emphasizing the importance of opening the mastoid in the direction of the course of the external auditory osseous canal.

FIGURE 29.

The mastoid opened showing how high the cells extend into the root of the zygoma.

FIGURE 30

Shows the modified radical, the type of operation frequently selected in special cases in an effort to effect the cure of a chronic suppurative process by drainage of the mastoid, antrum and epitympanicum, yet preserving and retaining in position the ossicles and membrana tympani. Note how superficial and far forward the bone covering the sigmoid portion of the lateral sinus. The cells in this case were of the diploetic type.

FIGURE 31

Represents the radical mastoid operation. This is selected as the operation of choice in the chronic form of middle ear and mastoid infection to rid, completely, all necrotic and infected areas from this region. The tympanum, the antrum and the mastoid are con-

verted into one large cavity. To accomplish this, it is necessary to remove the posterior part of the external osseous auditory canal, the facial nerve passes under it. (Fig. 32.) The fenestras ovalis and rotunda are clearly exposed. The pyramid showing the superior, posterior and external canals is seen above.

Note—To visualize clearly those structures lying within the danger zone, this dissection has been made more extensive than is usually performed on the living subject.

FIGURE 32.

The radical mastoid operation is extended in order to expose the semicircular canals and the course of the facial nerve, as it passes between the fenestra ovalis and the horizontal canal.

FIGURE 33

Shows the labyrinth opened. The instrument is shown passing into the cochlea under and back of the horizontal portion of the fallopian canal and protruding above the horizontal semicircular canal. The opening of the superior and posterior semicircular canals is seen just above the point of the instrument.

PLATE 1. FIGURE 12.

Partly sclerosed mastoid. A few small cells remain at upper posterior angle. Lateral sinus large, superficial, and far forward.

PLATE 2. FIGURE 13.

Small cell type of mastoid. Cell area very large, and extends to near angle of orbit. Normal.

PLATE 3. FIGURE 14.

Small cell type. Very small cell area. Normal.

PLATE 4. FIGURE 15.

Above—large cell type. Normal on both sides.
Below—A. Dense sclerosis. Lateral sinus well posterior.
B. Small cell type, normal.

PLATE 5. 7079R. FIGURE 16.

Type doubtful, either undeveloped or cells were obliterated early in life. Very little evidence of sclerosis. Lateral sinus fairly well posterior, but superficial.

PLATE 6. 7079L. FIGURE 17.

As compared with R, there is more evidence of sclerosis. There are a few cloudy cells toward the tip.

PLATE 7. 3284L. FIGURE 18.

Small cell type mastoid fairly large area. Cell shadows are cloudy. Slight sclerosis at tip. Lateral sinus rather far forward. 6881L shows this same mastoid 3½ years later. Note increase in sclerosis.

PLATE 8. 6881L. FIGURE 19.

Partial sclerosis with cloudiness of remaining cells. Note extent of cells posterior to lateral sinus which is rather far forward, sub-acute. Compare with 6881R, and also with 3284L, which is the same mastoid 3½ years previously.

PLATE 9. 6881R. FIGURE 20.

Small cell type, fairly large area. Cells extend forward to level with posterior border of temporomandibular articulation. Normal.

PLATE 10. 6220L. FIGURE 21.

Mixed cell type, small cell area. Normal.

PLATE 11. 6220R. FIGURE 22.

Sclerotic mastoid. Note bone defect in antrum region, and along anterior border of the lateral sinus toward the mastoid tip. Lateral sinus fairly forward and very superficial. Compare with 6220L for difference in appearance of groove for lateral sinus.

PLATE 12. 4001L. FIGURE 23.

Same cell area as shown on the right side. These cells are cloudy, showing less contrast. Earliest X-ray signs of acute mastoiditis.

PLATE 13. 4001R. FIGURE 24.

Small cell type, rather large cell area. Note cells extending forward over the zygoma. Normal. Compare with 4001L.

PLATE 14. 6912R. FIGURE 25.

Sclerotic mastoid, irregular, clouded cell structure posterior to antrum region and along anterior border of the lateral sinus toward the tip of the mastoid. Circular bone defect in skull about $\frac{5}{8}$-inch in diameter. In the absence of history of operation this probably indicates a necrotic area. Lateral sinus fairly well anterior.

Figure 1.

Figure 2.

Figure 3.

Figure 4.

Figure 5 (A).

Figure 5 (B).

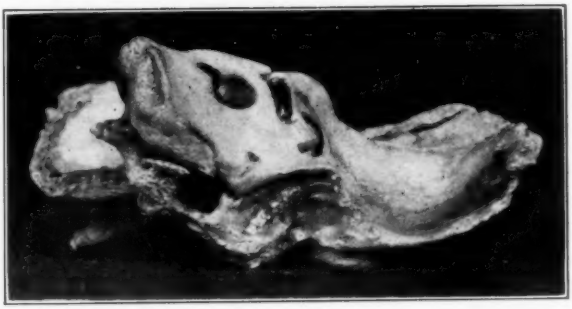

Figure 5 (C).

Figure 5 (D).

Figure 6.

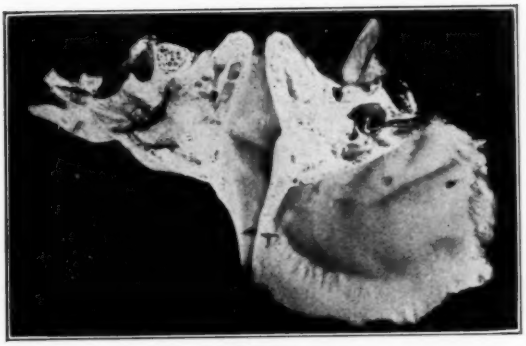

Figure 7.

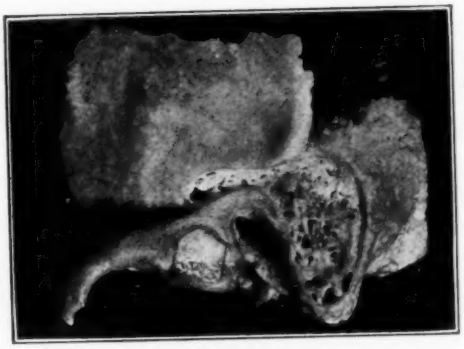

Figure 8 (A).

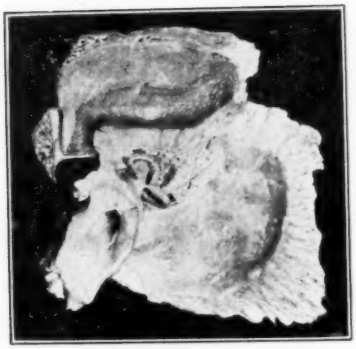

Figure 8 (B).

Figure 8 (C).

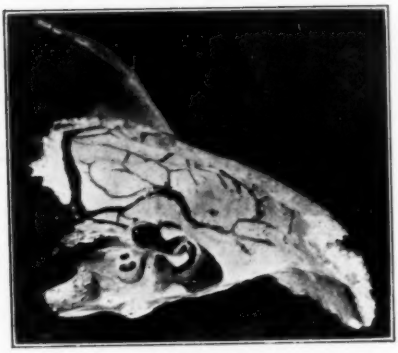

Figure 9.

Figure 10.

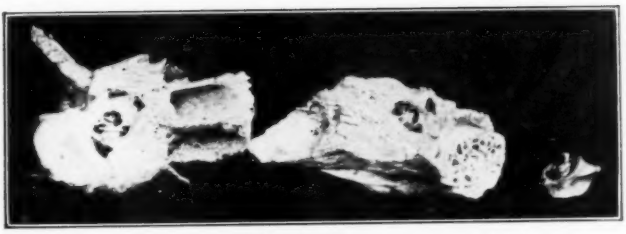

Figure 11.

Figure 12 (a).

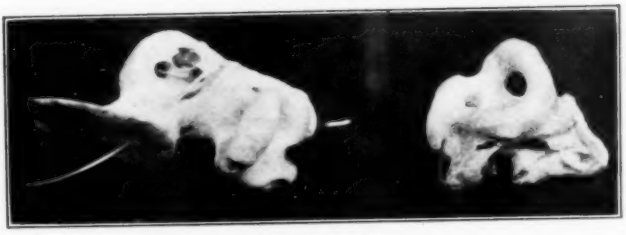

Figure 12 (b).

Figure 26.

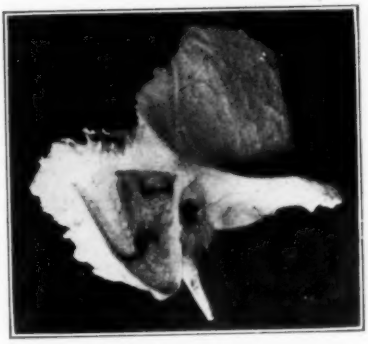

Figure 27.

Figure 28.

Figure 29.

Figure 30.

Figure 31.

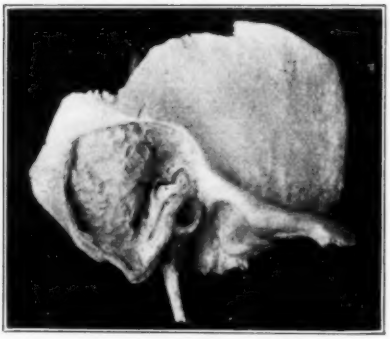

Figure 32.

Figure 33.

Plate 3. Figure 14.

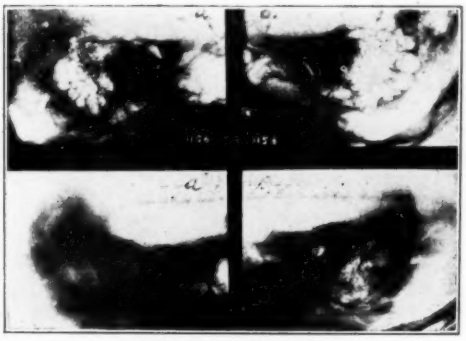

Plate 4. Figure 15.

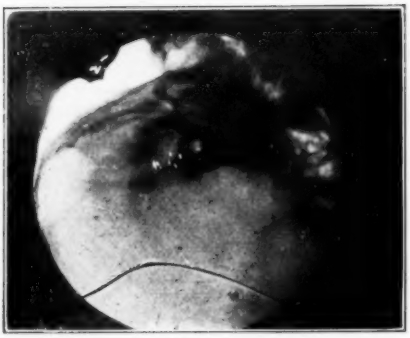

Plate 1. Figure 12.

Plate 2. Figure 13.

Plate 5. Figure 16.

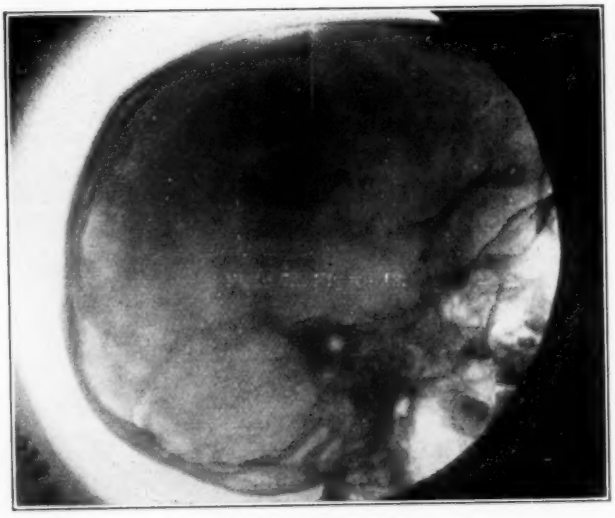

Plate 6. Figure 17.

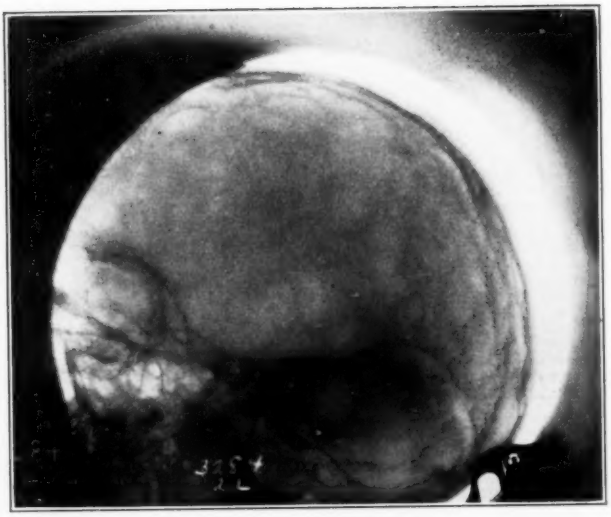

Plate 7. Figure 18.

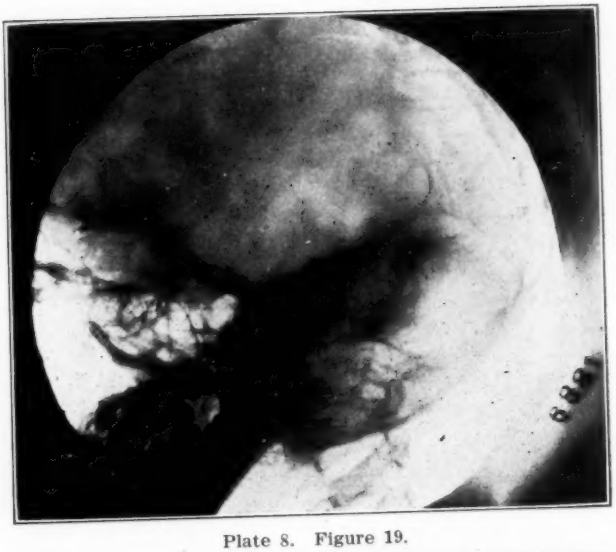

Plate 8. Figure 19.

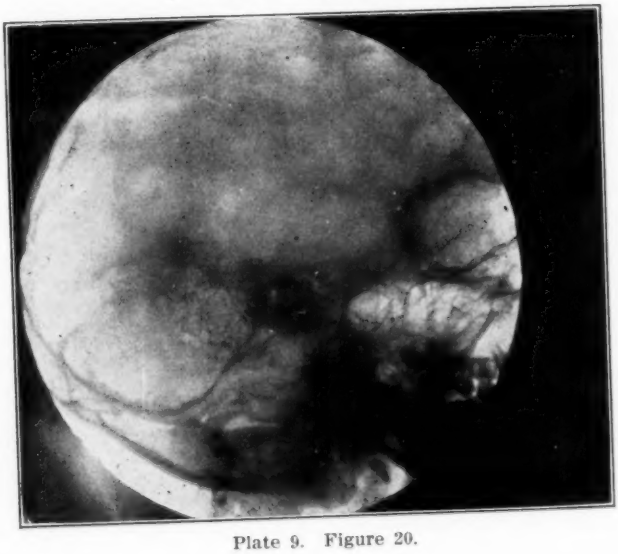

Plate 9. Figure 20.

Plate 10. Figure 21.

Plate 11. Figure 22.

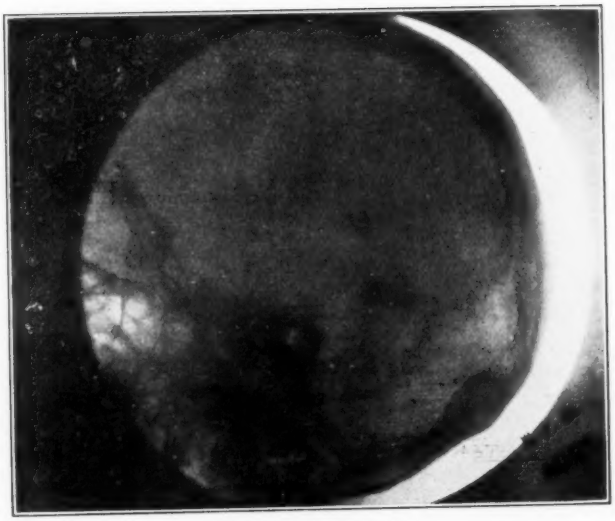

Plate 12. Figure 23.

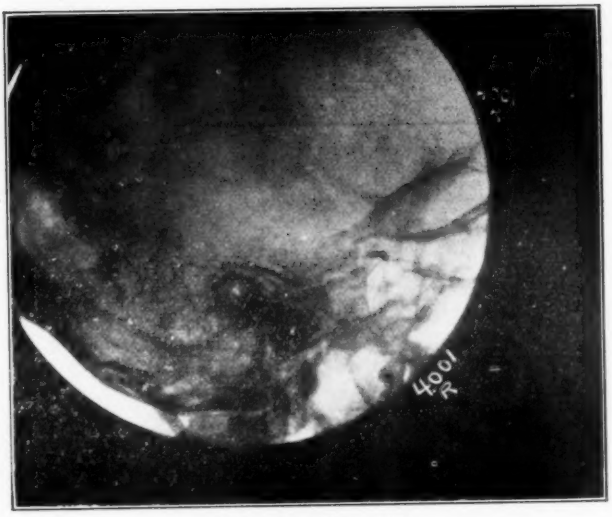

Plate 13. Figure 24.

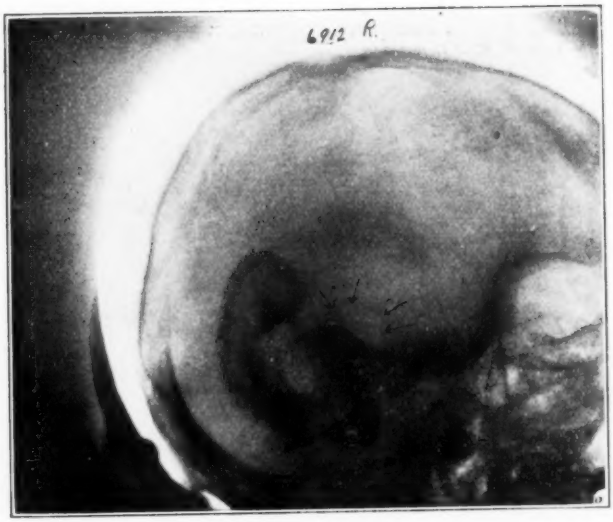

Plate 14. Figure 25.

XLVI.

SPHENOETHMOID SINUSITIS.

By John J. Shea, M. A., M. D.,

Memphis.

Wright,[1] in his History of Rhinology and Laryngology, relates of a slab which was erected in honor of the private physician of King Sahura, who ruled the Egyptians in 3500 B. C. From the translations it appears the physician cured a nasal infection of the king, so the king honored the ancient rhinologist and his wife by having the royal stone cutter inscribe the deed, so future generations might appreciate that the nares of even an Egyptian king could be pathologic, and that rhinologists of his day had adequate knowledge and were sufficiently versed in their art to cure the malady.

From ancient time until the present, acute and chronic infections have affected the upper respiratory tract. Formerly they were called rhinitis, and we were taught the symptoms and treatments of hypertrophic rhinitis, and of atrophic rhinitis, and of rhinorrhea, but today we appreciate that the membrane of the nares is the show window for the sinuses, and every rhinologic examination has as its fundamental basis a study of the accessory sinuses. The recent epidemic of influenza was thought by many to have been primarily a sinus infection, and subsequently a septicemia. Whether this deduction is correct or not, we are seeing five cases of sinus infection now, where we saw one of the disease prior to the epidemic.

Not all of the cases give a concise history of having begun with the "flu," but it is nearly universal for them to relate the influence the infection had upon their disease. The disease is found in three stages: acute, subacute, and chronic. The acute stage can be treated medically, and the subacute will often subside under medical treatment, but the chronic stage is seldom more than improved by anything less than an exenteration of the ethmoids and a drainage of the sphenoid.

*Candidates' Thesis for the American Laryngological, Rhinological and Otological Society, accepted January, 1922.

The use of radium for the relief of the postoperative headache when it is severe, and its application for the removal of the excessive granulations and recurring polyps is worthy of presentation. The ethmoids may act as a focus of infection and either directly or indirectly cause an inflammation of the optic or auditory nerve or both. The chief complaint of these patients is not always nasal, but may range from progressive loss of vision or hearing to a persistent cough with a loss of appetite and fear of tuberculosis. Mental symptoms are very common.

ANATOMY.

The ethmoid and sphenoid sinuses are so closely articulated as to be considered one structure. The line of demarcation between the anterior and the posterior ethmoid cells cannot be made out surgically, and in a great number of noses the relationship of the posterior ethmoid cells and the sphenoid is so complex that a complete exenteration of the ethmoids will require the removal of pars nasalis and the pars ethmoidalis.

The ethmoidosphenoid sinuses constitute a series of cells of varying communications formed by the ethmoid, the frontal and sphenoid bones, and limited by the superior maxillary, lacrimal and palate bones. The middle turbinate and the rudimentary superior turbinate are a part of the ethmoid bone.

The relationship of the sphenoid to the posterior ethmoid cells is not constant. In the majority of cases the sphenoid articulates anterolaterally with the posterior ethmoid cells, but there are many instances where the cells will surround the sphenoid superiorly.

The size of the middle turbinate is often dependent upon the condition of the ethmoids, and in those cases where the bulla has hypertrophied to the cystic state the middle turbinate will be displaced and moulded between it and the septum, and at times atrophy. The posterior end of the middle turbinate bears a constant relationship to the sphenoid sinus and can be relied upon as a landmark for the puncturing of the sinus.

The sphenopalatine and the anterior and posterior ethmoid arteries supply the ethmoid and the sphenoid, and the location of the sphenopalatine artery should be kept constantly in

mind while working in the sphenoid region, for injury to it will promote free hemorrhage and handicap the procedures.

The sphenopalatine artery is the terminal branch of the internal maxillary, and enters the nasal fossæ through the sphenopalatine foramen. The sphenopalatine foramen is formed by the articulation of the palate bone and the under surface of the body of the sphenoid, and transmits the sphenopalatine artery and the nasopalatine and superior nasal nerves.

The nerve supply is from Meckel's ganglion and the nasal branch of the ophthalmic; therefore block anesthesia is applicable in this field.

Mullins,[2] of Colorado Springs, worked out the lymphatic supply of the ethmoids as follows: "The lymphatic drainage is by way of the submaxillary and deep cervical and the retropharyngeal glands to the cervical lymph ducts, then into the great veins to the right side of the heart, and the pulmonary artery conveys the drainage to the lungs."

There is a type of ethmoids which may be termed "persistent infantile," inasmuch as it represents the undeveloped variety. The cells are small and few, with thick walls. The frontal sinuses are small or rudimentary, but occasionally there is a supraorbital ethmoid cell larger than the frontal, but, as a rule, in this type there are no atypical cells.

The morphology of the ethmoidosphenoidal cells varies to such an extent that we find cells which extend across the orbit for the full width of the frontal sinus, while at other times they encircle the sphenoid sinus superiorly or extend down into the pterygoid process. Similar to the mastoid process, there are times when it would be impossible to exenterate all the cells.

The sphenoid sinus occupies the body of the sphenoid bone and has six walls:

1. The anterior wall or face of the sphenoid, which is divided into the pars nasalis, in which is the ostium and the pars ethmoidalis, which articulates with the ethmoid bone completing the posterior cells.

2. The inferior wall, which forms the vault of the nasopharynx.

3. The posterior wall, which is the thickest and which is composed of the cancellous bone of the sphenoid.

4. The superior wall, which is the floor of the sella turcica. This wall is in direct contact with the optic nerves and optic chiasm. The dura mater reduplicates itself and stretches over the sella in such a manner as to make this space extradural. Hence, accidental puncture through the superior wall is not lethal, due to its being extradural.

5. The external wall, which is the thinnest and is in direct contact with the cavernous sinus. (Case 3.)

6. The septal wall, which may be deviated. The two sinuses are seldom of equal size. The cavity is lined by a very thin membrane which strips readily.

ETIOLOGY.

Age.—Dean[3] has called attention to the number of ethmoid and sphenoid cases found in children. No age appears to be exempt, each favoring the variety corresponding to its activities —that is, in children the usual inflammatory diseases are acute, so in sinusitis we are more apt to find an acute than a chronic sinusitis in children. In children we see the acute or subacute with active symptoms. Dean[3] reports a case found at autopsy in a child sixteen months old. The sphenoid, though present at birth, is less of an anatomic entity than the ethmoids, but after the fifth year it assumes its proper importance. In the young adult we see the above and a few of the atrophic varieties, while in the adult and the aged we see the acute, the hyperplastic and the chronic suppurative with its polyps and purulent discharge. The cases in the children act as foci of infection, while in the adult their effect is mostly direct.

Sex.—Males are slightly in the majority, due to their greater exposure and less care during the acute stages.

Social Status.—In the clinics the cases appear to be more severe than those met with in private practice, but this may be due to the fact that the clinic cases wait longer before they consult the surgeon.

Bacteriologic.—In turn, all of the pathogenic bacteria have been accused of being the cause of acute infection of the upper respiratory tract, and now Foster[4] claims a filterable virus to be the exciting cause of the acute cases. This confusion of knowledge is the reason for the failure of vaccines receiving a better welcome. But whatever was the active organism of

the late influenza epidemic, it seemed to be very potent in activating an infection in the sinuses. Cultures from the acute cases vary from day to day, and those obtained in the chronic cases may be from a secondary infection.

Protein Sensitization.—Protein sensitization has also attracted attention because of its influence in these cases. By constant stagnation of the circulation the reparative processes are retarded and metabolic changes occur in the parts, which promote chronicity.

Internal Secretions.—Internal secretions act similar to protein sensitization by their action on the circulation of the lining membrane of the nares and sinuses.

Trauma.—In the service at Base Hospital No. 57, only one case of bullet wound of the ethmoid was seen. This was a negro private who had been hit by a machine gun bullet, which passed through both nares and a secondary suppurative ethmoiditis developed. There have been cases of accidental puncture of the ethmoids, but the percentage is very small.

Extension.—An empyema of the maxillary antrum may ascend into the ethmoid and develop an acute ethmoiditis, though the reverse of this is usually the case. Similarly the sphenoid sinus may be infected from the ethmoids.

Diseases.—Influenza appears to have a predilection for infecting the ethmoid and the sphenoid, and the present increase of the number of sinus diseases may be attributable to the recent epidemics. Erysipelas can be the result of a chronic ethmoid infection and not the cause as formerly thought. (Case 2.) The acute exanthemata are complicated by infections of the sinuses and are the chief causes of the disease in children. Syphilis, tuberculosis and malignancy may either directly or by extension cause an infection of the sinuses. (Case 18, 19, 20.)

Climate.—Climate influences the course of the infection. The worst climate is a cold, wet, heavy atmosphere with rapid changes, and the best climate is one in which there is plenty of sunshine, but with enough rainfall to keep vegetation fresh and the atmosphere bracing. The extreme tropical temperature promotes atrophic degeneration. (Cases 4 and 5.) A change of climate will stimulate the nasal circulation, and the cases acquired on the coast will improve in the mountains,

while those contracted in the mountains will improve at the seashore. Sunshine will stimulate the healing during convalescence, and the acute cases are always worse after the sun goes down.

Hypertrophy of the Tonsils and Adenoids.—The hypertrophied adenoid and tonsil affect the ethmoids and sphenoid in three ways: (1) Mechanically, by blocking the discharge from the sinuses and causing stagnation, and in turn the collected discharge irritates the adenoid and keeps it in a constant state of inflammation. (2) There is a lymphatic connection which serves to spread an infection from the lymphoid tissue of the Waldeyer ring to the sinuses; and (3) by direct extension of the inflammation.

PATHOLOGY.

The ethmoid and sphenoid sinuses, being lined by mucous membrane, are subject to all of the forms of inflammation that mucous membrane present, and because of the membrane's construction, blending as it does with the periosteum, undergoes several peculiar pathologic changes.

In the acute cases there is an active inflammatory reaction of the membrane with a free flow of discharge. At first the discharge is an excess of the normal secretion, but as the disease advances, more pus cells are thrown out and the character of the discharge changes to mucopurulent or purulent. The congestion and discharge soon obstruct the ventilation and drainage of the cells, and absorption of the poison is manifested by constitutional symptoms, as fever and malaise. Under treatment the congestion is relieved and ventilation and drainage restored with a lessening of the purulent character of the discharge and of the headache. But if the infection is allowed to continue, the end result will depend upon the virulence of the infecting organism. In mild cases an immunity is soon developed and the inflammation subsides. This may be completely or with a remaining focus subject to reinfection. The severe cases completely obstruct the drainage, and the infection passes through the bony wall, causing a periostitis of the ethmoid orbital wall. If a perforation results, orbital cellulitis with its train of symptoms will follow. The frontal sinus may become blocked and infected or the

antrum utilized as a reservoir and an empyema of the maxillary sinus complicate the situation.

From infancy to the grave the membranes of the sinuses are constantly being abused, and the chronic conditions are brought about by the summation of the acute infections. In the cases where a focus of infection remains, the mucoperiosteal membrane is constantly congested, which interferes with growth and repair, and in time the bony walls of the cells become like paper and the ethmoids undergo cystic changes. There is an influence, the exact nature of which is not thoroughly understood, exerted by the endocrinal secretions. Theorizing, it may be that during the systemic stage the toxin influences the internal secretions and they in turn are deficient during the reparative stage of the inflammation, and in this way the atrophic condition of the bony walls develops.

In persistent infantile ethmoids the inflammation is not so active, but is more painful and more likely to become chronic. The normal pneumatic structure of the ethmoids allows a greater extension of the infection, with less probability of complication and chronicity, than the sclerotic ethmoid with its thick walls and small cells.

Extension of the infection to the bone will cause an acute osteomyelitis. This is more common in children than in adults and more common in the acute stage than in the chronic stage.

Briefly, the stages of pathology of the ethmoids are: (a) Acute, either catarrhal or purulent; (b) chronic, usually purulent; (c) atrophic and cystic; and (d) hyperplastic. The difference in the last two appears to be due to the character of the reparative process assumed by nature in its attempt to eradicate the disease.

The sphenoid sinus differs in its behavior towards infection, due to the fact that it occupies the center of a more massive bone, and is more abundantly supplied with nutrition. The acute and chronic suppurative changes occur after the same fashion, but cystic changes are never seen, though the thinness of the anterior wall at times is presumption that it too undergoes paperization. Where there is a marked difference in the size of the two sphenoid cavities and they both are infected, the smaller of the two will require a longer postoperative period to heal or will more readily become chronic. The an-

terior wall of the sphenoid springs from cancellous bone and is prone to produce excessive granulations at its base, which will close in any surgical opening unless care is taken to retard it and allow the epithelium to heal over the edge of the wound.

Whether polyps spring from the superficial layer and by absorption cause the underlying bony changes, or whether the bony change occurs first and the polyps result therefrom, is a mooted question, but today the opinion is that the polyps begin in the mucosa. Nevertheless as long as polyps reform, there is present pathology in the bone, and removal of their base will be necessary for a cure.

The lymphoid tissue of the pharynx and the larynx undergoes hypertrophy and congestion, due to the irritation from the discharge and as a part of nature's fight against the infection. The mouth of the eustachian tube may become obstructed and a eustachian salpingitis develop. An inflammatory hypertrophy of the subepithelial lymphoid tissue, constituting the lateral pharyngeal wall, and the posterior pillar of the tonsil, signifies a postnasal discharge and is convicting evidence of sinus disease.

CHRONICITY.

Each succeeding acute infection interferes with the nutrition of the membrane of the sinus and its bony framework, and through faulty metabolism the process of repair is weakened. The cells may become cystic or the membrane undergo polypoid degeneration. For the tissue to atrophy the destruction must be greater than the repair, and there must be a lessening of circulation. Here the role of the endocrins is brought into play. The persistent infantile type of ethmoid will, like its prototype, the sclerotic mastoid, readily become chronic. The pneumatic nature of the ethmoids permits of severe infections, with vicious inflammation, without loss of tissue.

CLINICAL HISTORY.

Chief Complaint.—The patient seldom gives as the chief complaint a symptom which is a direct accusation of the ethmoidosphenoidal sinuses. Because the discharge descends from the posterior nares into the nasopharynx and down to the larynx, the patient's attention is centered upon these regions,

and he complains of a clearing of the throat and an irritable pharynx or a productive cough, with or without hoarseness. If there is a marked hypertrophy of the ethmoids or the turbinates, blockage of nasal ventilation will be the principal symptom. White[5], of Boston, has attracted our attention to seeking a cause in the ethmoid for progressive loss of vision.

Likewise, we are constantly meeting cases of progressive deafness, which have as their cause an infection of the ethmoid or sphenoid sinuses, and whether the aural condition is due to extension of the infection through the tube or by the action of the toxins on the eighth cranial nerve is difficult to decide. (Case 29.)

When the sinuses are acting as foci of infection, the patient may complain of many and varied symptoms. This is best exemplified by the arthritis of childhood, as admirably pointed out by Dean[3] and his colleagues.

The west is full of patients who have been urged to seek a better climate before their pulmonary condition advanced beyond repair. They had sought relief from a cough that was productive upon arising in the morning, loss of ambition, increasing lassitude, and an afternoon elevation of temperature of a degree or so. This train of symptoms attracted the attention of the diagnostician towards their chests, and he, failing to find any physical signs, had their lungs radiographed, with the usual findings, "peribronchial thickening, mediastinal cloudiness, and a few apical scars." During all this time the case is still having a postnasal discharge, and each morning finds the discharge about the larynx ready to be expelled. Hence the diagnostician, in despair, advises "Go west," instead of sending them to a competent rhinologist. (Cases 6, 7, 8, 9, and 10.)

Headaches.—The typical headache of sphenoid sinusitis is in the suboccipital region or deep in the center of the head, radiating to the surface at the vertex. The anterior ethmoids will cause a headache through the temples and over the eyes, with pain on using the adductor muscles of the eye. The posterior ethmoids, by their influence on Meckel's ganglion, may cause pain in the various distributions of the ganglion, but the most notable point being two inches posterior to the external auditory canal (Sluder[6]). There is a depression of

cerebration in these cases and the memory suffers. Concentration is difficult, but all can be cleared away by removal of the cause. (Cases 2, 3, and 28.)

PROGRESS.

During the acute period, discharge and interference with nasal ventilation constitute the chief complaints of the patient, while during the subacute stage the obstruction will be greater or less, depending upon whether the cells hypertrophy or atrophy. The discharge at first remains mucoid in character, but greater in amount, becoming mucopurulent and finally purulent. If the acute infection is caused by the hemolytic streptococcus, the discharge will be tinged with blood. (Case 1.) The subacute stage may last throughout a winter and with spring the symptoms and signs decrease, completely to a cure or incompletely to the chronic stage. The lack of reaction marks the chronic stage, and the progress becomes retrogressive without inflammatory reaction, except whenever there is an acute exacerbation. With each of these acute exacerbations the chronic condition becomes worse, until the atrophic or cystic stage is reached. It is difficult to express a prognosis on whether or not the tissues are capable of regeneration. When least expected the tissues make a favorable postoperative recovery, and in the end healthy membrane may be seen where before were scabs and an atrophic membrane.

DIAGNOSIS.

The diagnosis of ethmoidosphenoid sinusitis required a thorough examination of the nares, transillumination, suction and a legible radiogram.

Inspection.—Occasionally there will be a dermatitis about the vestibule of the nose from the irritation of the discharge, especially in children. The condition of the membrane of the septum and the turbinates will range from an active congestion to a boggy edematous infiltration. During the acute stage all erectile tissues will be congested, while in a number of chronic cases the membrane will demonstrate that there is a deficiency of vasomotor control. Discharge may be seen coming from under the middle turbinate or posteriorly in the choana above the inferior turbinate. If this is wiped away and shortly returns in the same region, then one can feel certain

that it is not being secreted by the exposed membrane, but is coming from the sinuses. Polyps generally denote sinus infection, and their position is a help in determining which sinus is diseased. Hypertrophy and congestion of the subepithelial lymphoid tissue of the pharynx are convincing evidence of a postnasal discharge, and the presence of the discharge in the nasopharynx is a verdict against the sinuses.

Transillumination.—Transillumination is better for studying the maxillary and the frontal than the ethmoid sinuses, but when these cavities illuminate clearly and the nares are darker than normal one may suspect the ethmoid cells of being filled with discharge or their membrane thickened by chronic inflammation.

Radiography.—To Law, of the Manhattan Eye and Ear Hospital, is due credit for perfecting a technic that will give a surgical picture of the sinuses. Following Law's technic, the ethmoid cells will be thrown free from the other shadows and will stand out in bold relief. The anteroposterior picture will display the maxillary, ethmoidal, frontal and sphenoidal sinuses, while the lateral picture will show the posterior ethmoid and sphenoid sinuses and the depth of this frontal sinus. Taking the ethmoid, the frontal and the orbit, the two sides should be compared, and in unilateral involvement the contrast will be marked. If the lateral picture shows the frontal to be of average depth, the anteroposterior picture in normal sinuses will show the frontal ventilated and not darker than the ethmoid cells, or lighter than the orbit. When the frontals are cloudy the ethmoids will be cloudy or hazy. There is a common picture of the frontal being of average size, but more cloudy than its fellow, or when the two sides are concerned both frontals being cloudy, but still the ethmoids appear well ventilated. The frontals should be no more cloudy than their corresponding ethmoids, and if they are it means that the ethmoids have advanced from the inflammatory to the cystic state. At operation this variety of ethmoids will show atrophy, and in the past the radiogram reading in these cases was "ethmoids appear well ventilated." The truth of the matter is that they are too well ventilated. Careful scrutiny of the ethmoids will show cystic changes. This is best illustrated when there is a supraorbital ethmoid cell well ventilated under a cloudy frontal

sinus. Discharge and thickened membrane will equally shadow the ethmoid region as shown by taking a picture before and after the use of vacuum treatment.

When the lateral picture shows the sphenoid to be of average size, it should be visible on the anteroposterior view as a heart-shaped object about half way up the nares. If its outline is blurred, a diagnosis may be made of thickened membrane or pus in the posterior cells and sphenoid sinus. Occasions will arise when the comparative measurements of the two sides will not determine whether or not the space under consideration is the sphenoid or an atypical posterior ethmoid cell, and the only positive means of deciding will be to take a lateral view with probes in the ostia of the sphenoids.

The adverse criticism of the value of the radiogram in sinus diagnosis has been due to the poor pictures rendered for interpretation. No diagnosis could be made from some of them, for the sinuses were not in surgical relationship and their contours distorted. By following the technic of Law, the details of the sinuses can be brought out in such a manner that reliable information may be obtained, and each surgeon should be able to read his own pictures and by studying the views after the operation develop a better knowledge of this most important adjunct. Formerly the only information desired by the surgeon was the size of the sinuses, but today we expect to obtain an inkling of the pathology and a guide as to the best surgical procedure.

Suction.—The use of vacuum properly applied will drain the sinuses of their discharge, and by observing its location and character valuable knowledge may be obtained. It is the constant discharge as shown by suction that convicts a sinus. If the discharge is to be studied bacteriologically the nose should be cleansed with normal saline, then suction applied to withdraw the discharge, after which a sterile glass tube should replace the nasal tip, and suck a specimen from under the middle turbinate or from the posterior naris.

TREATMENT.

Rest is the most important therapeutic agent in the acute and the subacute stages. A patient is always better after a good night's rest.

Acute Stage.—Not all suppurative inflammation of the ethmoidosphenoidal group requires operation. Many of the acute cases subside of themselves and others can be hastened by promoting better ventilation and drainage. The use of an adrenalin spray with a 1/4 per cent cocain solution as the basis will shrink the congested turbinates and the membrane lining the septum. The judicious application of suction will aid the drainage, and the removal of the discharge from the nares by a saline douche will give comfort to the patient. The patient can be furnished a water suction device of the Brawley or Gray type for home use, and also be taught how to irrigate the nares without danger to his ears. The headache of the acute cases will be relieved by ventilating the sinuses with the above treatment, and in those obstinate cases with severe headache 5 mm. of pituitrin extract (surgical) hypodermically will constrict the vessels and open the way to ventilation. If this plan is followed during the first day of the socalled influenza cases there is less absorption of toxins and the case will assume the characteristics of the "three days' fever" malady. An oil spray of guaiacol will serve well as an emollient to the washed membrane, and also act as an antiphlegmatic.

Subacute Stages.—Before advising an operation the above treatment should be ordered for daily use at home. Sustaining treatment and the administration of autogenous vaccine will aid. If there is a deflected septum or a hypertrophied turbinate, or both, the correction of these deformities will afford better ventilation and drainage. The ethmoid is so constructed as to handle infections if given an equal chance. There is a future for endocrine therapy in these cases, for some improve under thyroid gland, gr. 1, twice a day.

Chronic State.—The above treatment will often be necessary to convince the patient that he is suffering from a more persistent and pernicious ailment than a "head cold" or "hay fever." The cleaner the cells are of discharge the less risk there will be of infection at operation and of postoperative fever. If a submucous resection of the septum is needed, it had better be done as a preliminary step, and there are some surgeons who will at this time amputate the middle turbinate, but the latter structure is a guide as to the situation of the cells and its posterior attachment a landmark for the punctur-

ing of the sphenoid sinus and had better remain until the exenteration of the cells is attempted.

Postoperative Stage.—Whether the intranasal or the radical operation is done, the postoperative care must be carefully managed and not delegated to an assistant. The restoration of the two functions of ventilation and drainage is necessary for a success. If there has been no packing the nose should be sprayed frequently with normal saline and an oil instilled to prevent the formation of scabs. Where packing is employed it should be kept moistened with saline solution and its removal effected as soon as possible, and at the end of twenty-four hours if feasible. The less handling of the parts until the granulations have become organized, the more rapid will be the improvement. When the granulations show signs of becoming excessive, steps should be taken toward their eradication. Silver nitrate or the actual cautery will destroy the hypertrophied granulations and allow the epithelium to cover over the exposed surface. If the granulations become very excessive or small polyps reform in spite of treatment the use of radium is advised.

When to begin the use of the suction will depend upon the discharge. If the discharge is blocking the passage and simple douching cannot remove it gentle suction may be tried.

The task of keeping the sphenoid sinus open is often difficult. The epithelium must cover over the edges of the opening or it will decrease in size until a new anterior wall has formed, thereby blocking the sinus. If the sphenoid sinus does close, dilatation of the opening with cotton tampons or its removal with the biting forceps will be necessary to effect a permanent opening. This may be repeated many times before it becomes permanent.

Radium.—The task of keeping granulations under control is difficult. Radium will serve better than any other measure. The granulations spring from the cut surface and serve as a supporting framework for the epithelium to grow over and heal the wound. If the granulations become too excessive the epithelium cannot surmount them. The experience in Case 23 showed two things: First, that radium will reduce excessive granulations and scar tissue in the ethmoids and

sphenoids, and secondly, that it will give more than temporary relief from the headache.

The radium by its action on the new formed tissue produces a fibrosis with a relief of the pressure. The nares are packed off and protected, except for the part to be treated, and the needles encased in wax and placed in apposition to the ethmoids and retained there by cotton plugs. The first dose is usually for one hour of 50 mg., and depending upon the reaction, the second dose is either increased or diminished or more thoroughly screened. Within two weeks there is marked improvement. We have asked of radium the impossible. and because it cannot perform a miracle we are disappointed, but its use in the removing of excessive granulations and polypoid degeneration of these wounds is possible and can be relied upon. The idea has been advanced that the radium tends to produce a fibrosis, and when this tissue is removed it will not return, whereas when the granulations are removed their recurrence is nearly certain. This is shown by treating a very pernicious reformation case with radium and then removing the excessive tissue.

OPERATION.

The question of the choice between the intranasal exenteration of the cells and the external radical operation will depend upon:

(a) The size of the sinuses; the larger sinuses with ramifications will demand the radical operation.

(b) The cases complicated by a chronic infection of the frontal sinus will be better treated by the radical method.

(c) Those cases in which the ethmoid cells are acting as a foci of infection will require a thorough exenteration as only the radical procedure affords.

(d) If the intranasal operation has been thoroughly done and the after treatment carefully managed, with no improvement in the symptoms, the radical is indicated.

The technic of the Lynch frontoethmoid operation is the best for the radical ethmoidosphenoidal procedure.

The best operative position for the patient is half reclining on a full length operating table.

Thorough anesthesia of the parts may be obtained by swabbing the surface of the turbinates, the ethmoids and sphenoids with a 4 per cent solution of cocain to which ten minims of adrenalin solution to the ounce has been added, and then injecting the region of Meckel's ganglion with a 2 per cent solution of novocain. The postnasal branch of the sphenopalatine may be injected on the anterior surface of the sphenoid, and a similar injection at the anterior attachment of the middle turbinate will anesthetize this region. By its action on the sympathetic element of the ganglion vasor constriction of the area is produced. In cases where there are many polyps and the ethmoids are markedly congested, the above will not control excessive bleeding, but if pituitrin be given intramuscularly throughout the operation, the field will be less sanguinous. Only two or three minims every fifteen minutes will be sufficient and will greatly expedite matters. Half an hour before the operation a hypnotic is administered, and if the necessity arises subsequent doses are given.

Before starting the operation a small piece of gauze is inserted deep into the nares against the inferior wall of the sphenoid sinuses. This prevents blood from dripping down into the throat, and thereby avoids the delay occasioned by the patient constantly clearing his throat.

The middle turbinate is thoroughly amputated by cutting its membranous attachment with a long nasal knife, and severing the bony attachment with turbinate scissors, either curved or straight, as the necessity demands. Then its removal can be accomplished with a nasal snare. There will remain a tag of varying size at the posterior attachment of the middle turbinate. Using this as a guide, the sphenoid sinus is entered by direct puncture with a sphenoid punch forceps. If the head is in a half reclining position the instrument is directed between the tag and the septum on a direct line. The sphenoid sinus will always be at a greater depth than the tag, and in this manner the tag can be utilized as a landmark in measuring. No attempt is made to locate and enter the ostium of the sphenoid. The beginner expects the sphenoid sinus to occupy a higher plane and is often tempted to puncture too high. After the first bite the resection of the pars nasalis is accomplished and then the pars ethmoidalis removed from

above downwards, care being taken to respect the sphenopalatine artery. The work on the sphenoid should be done first, as it is the most delicate, and a clean field will allow of a greater precision. Next should be the removal of the posterior ethmoid cells, which articulate with the sphenoid. If there are any cells which extend above or externally to the sphenoid they should be cleaned out. All of the older methods worked from before backwards, and by the time the posterior ethmoid cells and the sphenoid were reached the operative field was so obscured as to detract from the exactness necessary for the success of the operation. Care can be taken not to wound the sphenopalatine artery, which lies in front of the external inferior angle of the anterior wall of the sinus. With a sharp biting forceps the ethmoid cells are removed from behind forward, starting on a plane with the severed attachment of the middle turbinate and working from behind forward and from above downward. The cells above the middle turbinate must be cared for properly with a small sharp biting forceps and not curetted. No tearing should occur; each grasp should acquire only what it is capable of removing by a clean bite. The anterior cells require a sharp angled forceps and at times a curette. The bulla and the membrane covering the uncinate process should be reduced thoroughly, or else the drainage of the anterior cells will be blocked during the convalescence. The cells are removed by sight and not by touch, leaving the membrane lining the remains of the cell walls, which are made up by the orbital plate. This is done with the hope of less granulations springing up from denuded bone. The cavity is flushed with ice cold normal saline solution, which will stop the capillary oozing and allow a thorough inspection of the field. Any remaining diseased tissue is removed and the frontal duct probed as to its patency, but no attempt is made to curette the lining membrane of the duct. If the hypertrophied cells about the duct are reduced the duct will drain and no stricture of it will occur from granulations, whereas, if an attempt is made to curette the duct granulating tissue will soon block the drainage of the frontal sinus.

Whether to pack or not depends on how dry the parts are at the finish of the operation. If there is free bleeding from the region of the sphenopalatine artery and crushing of the

parts with an artery forceps fails to stop it, not to pack is a risk. When packing is used, gauze soaked in liquid petrolatum to which has been added tincture of benzoin compound is preferable. The distal end is anchored in the sphenoid cavity and allowed to drop down to the soft palate and out along the floor of the nose to the vestibule, where it is fixed by the assistant, and into this sack the remainder of the packing is inserted. The packing should not be allowed to dry, but should be kept moistened with either more of the oil or normal saline solution. This will aid its removal. The disadvantage of packing is the possibility of damming up infection and of hemorrhage upon its removal. The earlier that the packing can be removed without fresh hemorrhage the better it will be for the patient and the more rapid will be the healing. If secondary hemorrhage occurs on the fourth or fifth day a postnasal pack will be more efficacious and less dangerous than the simple anterior pack.

The use of suction during operation is as valuable as in throat work. At first the author used an ordinary glass drinking tube, attached to the suction part of the ether suction apparatus, but now he uses a specially constructed metal tube, capable of being boiled. Suction will expedite matters by preventing the loss of time utilized in sponging or by the patient clearing his throat.

RADICAL OPERATION.

The incision of the Lynch operation is of value because of the exposure it affords. The frontal sinus is attacked and obliterated and the ethmoid cells removed by sight. The sphenoid is readily exposed and its anterior wall removed, and if it contains polyps the lining membrane is removed, but if it is lined by a thin clean membrane this is left. The complete removal of the cells will eliminate the ethmoids as a focus of infection.

COMPLICATIONS.

The complications are of the disease and of the operative treatment. Those of the disease are due to extension of the infection and depend upon the route of the extension. If the infection perforates into the orbit there will occur an acute orbital cellulitis with its possibility. The eye may be involved

from the sinus infection in the following manner: ulcer of the cornea, phlyctenular conjunctivitis, uveitis, retrobulbar neuritis, chorioretinitis, panophthalmitis and orbital abscess. An acute osteomyelitis is more common in children and acute inflammation than in adults and chronic inflammation. Mullins[2] has called attention to the number of pulmonary conditions due to ethmoid and sphenoid infection, and in children we see an inflammation of the lymphoid tissue of the pharynx and of the membrane of the larynx. Dean[3] illustrates with case histories the relationship between multiple arthritis in children and paranasal sinus disease.

Meningitis or extradural abscess is possible, but seldom seen. A general septicemia (Case 1) is more common than usually realized, and many of the fatalities in the influenza epidemics were of this type. The surgical complications are as follows:

Anesthetic.—The committee upon local anesthesia of the American Medical Association has awakened a true appreciation of the dangers of this form of anesthesia. The administration of the preliminary hypnotic reduces the dosage of cocain or its allied drugs, and also prepares the nervous system for the ordeal of the operation. Hurd,[7] in his paper on scopolamin at the Boston meeting of the A. M. A., 1921, endeavored to prove that shock entered into the socalled cocain poisoning and that the administration of a preliminary hypnotic reduced the possibility of shock. This was refuted by Mayer on the grounds that of the cases reported the symptoms pointed to poisoning and not to shock. Care must be taken in the use of cocain or its allied product, and each case is a rule unto itself. If poisoning does occur, the administration of ether is the most efficacious remedy. Sustain the heart, as the depression is most acute upon the cardiac center. By block anesthesia less drug is necessary and the risk is reduced. Accidents have been reported due to cocainization of the larynx from the too free use of cocain in the posterior nares, thereby abolishing the laryngeal reflexes and allowing obstructing clot to enter the glottis.

Hemorrhages.—Primary hemorrhage may occur from the anterior ethmoid artery or the nasopalatine. The former may be stopped with bone wax, and Yankauer[8] has advised crushing

the latter with a long artery forceps at its entrance into the nasal fossæ. If the bleeding is moderate a little patience will often allow the formation of a clot. When a light pack will not control the bleeding a postnasal tampon should be used.

The use of an adrenalin spray every fifteen minutes for the first three hours and every half hour for the remainder of the first half day will aid in the prevention of late primary hemorrhage. After the oozing has increased to a flow and the nares and nasopharynx have been filled with an unorganized clot, the spray will be of no value and packing will be necessary. Ice compresses of gauze packs constantly applied will supplement the adrenalin spray.

Secondary hemorrhages are most frequent on the fifth day and may occur as late as the tenth day. If simple remedies, such as ice compresses, irrigating the nares with ice cold normal solution or adrenalin spray do not control the bleeding, the administration of morphin, gr. 1/4, and 5 cc. of hemostatic serum should be tried. The flow down the pharynx is the indicator as to the severity of the hemorrhage and should be closely watched. If packing is to be resorted to, the postnasal tampon is the most efficacious, and will cause less absorption of toxins. Infection with a resulting slough causes secondary hemorrhages by opening greater vessels.

Surgical Accidents.—If all the work is done by sight and not by touch, and each bite removed cleanly, there will be fewer surgical mistakes. The beginner expects the sphenoid to be situated too high, and persistent attempts at entrance of the cavity superiorly will perforate the floor of the frontal fossæ. The puncture of the posterior wall of the sphenoid is difficult but possible. If the sella turcica is perforated the danger of infection is the only serious worry, and the formation of its dural canopy minimizes this possibility. If there is an escape of cerebrospinal fluid the puncture will be irrigated and infection is less probable, and when there has been sufficient loss of fluid the pressure will be reduced and the wound will close. Perforation of the orbital wall is more apt to occur when an attempt to puncture through into the antrum is tried. Of course the removal of a cell whose wall is formed by the lamina papyracea exposed the orbital cavity.

Infection.—The less packing inserted the less likelihood of infection, and of all the complications infection is the most to be feared. The majority of intracranial complications are due to the spreading of the infection by the lymphatic and the veins. As above stated, secondary hemorrhages are from infection and in a measure tend to lessen the infection. Careless puncturing through into the cranial cavity will carry along contamination and produce meningitis or abscesses.

Meningitis is as a rule acute and fatal, but extradural abscesses may be subacute or chronic, and under proper treatment drain and effect a cure. There are times when the headache increases, the temperature rises a degree or so, and the neck muscles tighten sufficiently to prevent the patient placing his chin upon his chest without suppuration developing within the cranium. These cases are irritation of the meninges and require immediate attention. If they are packed, removal of the packing is imperative, and if they are not packed, shrinkage of the stuffy membrane by the use of the adrenalin spray or pituitrin hypodermically will promote ventilation and drainage. In twenty-four or thirty-six hours the headache should improve, and by the second day the rigidity of the neck muscles should be less. If there are no improvements by this time the case should be studied from a neurologic standpoint, to ascertain if the infection has spread inwards to the meninges.

Excessive Granulations and Reformation of Polypoid Tissues.—The rapid development of obstruction to the ventilation and drainage of the sinuses by the reformation of polypoid tissue or excessive granulations does not always denote necrotic tissue. This is proven by the removal of this new formed tissue with radium. If there was necrotic bone the simple application of radium would not obliterate it The excessive granulations are formed because the epithelium cannot grow inward from the edge as fast as the granulations can spring from the bone. The presence of excessive granulations or polypoid tissue will produce a return of the discharge and in time a recurrence of the symptoms.

Etiology.—Case 1 illustrates that ethmoid sinusitis could be the beginning of an influenza infection. Case 2 illustrates that erysipelas may have its focus in the ethmoids.

Anatomy.—Case 3 demonstrated how close the internal carotid can be to the lateral walls of the sphenoid.

Climate.—Case 4 illustrates that the tropics tends to produce atrophic changes. Case 5, same as 4.

Pulmonary.—Cases 6, 7, 8, 9 and 10 illustrate that the chief complaints may be pulmonary symptoms.

Extension from the Antrum.—Cases 11 and 12 illustrate that the ethmoids can be involved by the extensions of the infection from the maxillary sinus.

Secondary Hemorrhage.—Cases 13 and 27 illustrate secondary hemorrhages.

Methods of Locating the Sphenoid Sinus.—Cases 14 and 15 illustrate the value of the X-ray in deciding the position of the sphenoid when there is doubt as to its location.

Ocular Manifestation.—Cases 13 and 16 illustrate the influence the ethmoids may have on the vision and the rapid improvement which follows their exenteration. Case 17 illustrates how much pathology may be in the ethmoids, as in this case, to even cause an exophthalmus without any effect upon the vision.

Neoplasms and Syphilis.—Case 18 illustrates a suspected malignancy, but the laboratory report of chronic inflammation was sustained by the subsequent history. Case 19 illustrates a malignancy of the ethmoids and maxillary antrum. Case 20 illustrates a gumma of the ethmoids.

Children.—Cases 10, 21 and 22 illustrate the condition as seen in children.

Radium.—Cases 23, 24, 25 and 26 illustrate the value of radium in the postoperative treatment where excessive granulations are a complication.

Mental Symptoms.—Cases 27 and 28 illustrate the inability of concentration as seen in the sphenoid infections.

Auditory.—Cases 29 and 30 illustrate the influence the ethmoids have on aural pathology.

Complication of Meningitis.—Case 32 shows an apparent hemolytic infection of the meninges.

CASE HISTORIES.

Case 1.—Sergt. B., admitted to Base Hospital No. 57 for an acute infection of upper respiratory tract. After being up

and about for two days, developed an acute left frontal headache, and by evening his temperature had risen to 102°. The following morning there was a beginning orbital inflammation with tenderness on pressure over the left frontal sinus. A septic temperature persisted in spite of intranasal drainage. On the fourth day a severe epistaxis from the left nares, which was followed by others, and in another twenty-four hours patient had vomited blood and coughed up fresh frothing blood. A blood culture was positive in the next twenty-four hours for hemolytic streptococci. A transfusion on the fifth day gave a new lease on life, and the temperature remained below 101° for the next day, but the improvement was only temporary, and the hemorrhage reappeared from all the mucous membranes. A second transfusion was of no avail and the patient died. At autopsy there was evidence of hemorrhage from the left ethmoid and a clot filled the frontoethmoidal region. Evidently the left ethmoid had been the site of the original hemolytic streptococcus infection. Free blood was found in the lungs, the stomach, the pericardial and pleural sacs, and under the capsules of the spleen and kidneys. With all the hemorrhages the tissues were not depleted. This case resembled in many respects the fulminating type of influenza. The local manifestation of infection in the left ethmoid and frontal sinuses, with the resulting orbital cellulitis, were the only differentiating features. With less local signs the case could have been diagnosed influenza, and I think many of the pernicious cases which died in twenty-four to forty-hours were of this same type.

Case 2.—S. E. M., age 36. Chief complaints were pain in left antrum, a productive cough, loss of weight and strength. For the past three years patient had been a tubercular suspect. During an acute exacerbation of the ethmoid infection patient developed a chill, temperature of 103.6, with a sense of fullness over the nose. By afternoon the blush of erysipelas was present over the usual nasal position, which brings up the question, "Are the ethmoids the focus of recurrent erysipelas which appears without a visible wound?" Examination: Nasal membranes were thin and coated with dried discharge and the radiogram showed the ethmoids to be cystic and the left antrum to be cloudy. The ethmoids were exenterated and the

sphenoid was thoroughly drained. Two openings were made into the left antrum, one below the inferior turbinate and one above, by removing the ethmoid cells about the natural opening. As the membranes granulated and healed, the discharge ceased and the cough subsided.

Case 3.—J. H. G., age 44. Chief complaints were asthma and a chronic nasal obstruction for years, for which a submucous resection of the septum was performed with amputation of the anterior half of the middle turbinates in 1911. Because of repeated acute infections his tonsils were removed. In 1919 examination showed the nasal membranes to be pale and edematous and a few polyps sprang from under the stumps of the middle turbinates. The ethmoid cells were hypertrophied and the septum still deflected high to the left. The radiogram revealed a cloudiness over the ethmoids, and the sphenoids were of average size. Operation: The right ethmoid cells were exenterated and the anterior wall of the sphenoid was resected. The left ethmoid cells were more difficult to approach, due to the deflection of the septum. Upon removal of the left sphenoethmoidal partition a pulsation was visible through the lateral sphenoid wall, and an erosion of this wall by the internal carotid was suspected but not proven. No packing was inserted. Convalescence was uneventful until the evening of the fifth day, when the patient complained of severe pains through the temples and pain upon moving the eyes. His temperature was 100 to 104 degrees and he was unable to lower his chin to his chest. Examination of the nose was negative, but a cathartic was administered and an adrenalin nasal spray ordered. The next day conditions were the same, but the seventh day saw improvement, and on the eighth day nasal ventilation was free and there were no further headaches, but the movement of the head caused a sense of restriction between the shoulders for several days.

Case 4.—Mrs. C. E. G., age 26. Chief complaints were frontal headache and a recent acute maxillary sinusitis. Ten years ago, while living in southern Louisiana, her nasal infection took on the nature of an ozena (a condition often met with in cases which go farther south, as into Central America). Examination showed the nasal membranes to be atrophic and the remains of the turbinates were bathed in a purulent

discharge. Suction was productive of more purulent discharges from the ethmoid region. The radiogram revealed a cloudiness of both ethmoid regions and an increased density over the left maxillary sinus. The frontal sinuses were clear and the sphenoid sinuses of average size. Operation: The right ethmoid cells were easily worked out, though they extended high, but the left cells had an abnormally firm bony capsule. This same condition held true with regard to the sphenoids, and only after careful measuring and by direct puncture was the left sphenoid entered. A persistent oozing from a high posterior ethmoid cell complicated affairs, and the region could not be worked out as thoroughly as the right side. This was productive of profuse granulations in this region and required energetic postoperative attention before thorough drainage was established.

Case 5.—E. W., age 11. Chief complaint was a chronic nasal infection, which had the history of an acute sinus infection while in the tropics that was complicated by an atrophic rhinitis. Examination showed the turbinates to be small, and there were many adhesions between the septum and the turbinates. At operation the adhesions were dissected and the turbinates made free. Wax plates were placed between the septum and the freed turbinates and kept there until the raw surface had healed. The postoperative treatment consisted of the daily use of suction and thorough nasal irrigation. These cases of acute sinus infection will become atrophic if the patient moves into the tropics.

Case 6.—Mrs. R. D. C., age 37. Chief complaints were obstructed nasal breathing, profuse purulent discharge, both anteriorly and posteriorly, and a persistent productive cough. Examination showed the ethmoids to be hypertrophied and that the discharge was coming from under the middle turbinates. Radiogram showed all the sinuses to be cloudy and the sphenoid sinuses of average size. Operation: A bilateral exenteration of the ethmoids and sphenoidotomy. The tissues were very congested and oozing was very profuse during the operation and required packing. Convalescence was uneventful, and as the granulations healed the discharge became less and the coughing stopped. This case had been studied for

years as to the cause of the coughing, but the postnasal discharge had never been considered.

Case 7.—McK., age 46. Chief complaints were obstructed nasal breathing, asthma and a persistent productive cough. Family history of one brother out West for tuberculosis. Examination showed nares filled with polyps to the very outlet. Radiogram revealed all the sinuses to be cloudy. Operation: A bilateral exenteration of the ethmoid cells and sphenoidotomy. There was free oozing during the operation and the nose was packed. The hemorrhage upon unpacking was so profuse as to require consideration of repacking. Convalescence was uneventful, and as the acute postoperative stage passed away the symptoms grew less, and inspection two months later showed the sinuses well drained and the coughing reduced to a minimum.

Case 8.—Mrs. T. W. G., age 30. Chief complaints were that in 1919 had influenza and since then a productive cough for which she had been sent to Texas as a suspected tubercular patient. She also complained of a frontal headache, worse on the left, and tenderness of the scalp over the left parietal region. Examination showed the nasal membrane to be congested and the turbinates jammed. The septum had a spur to the left. Transillumination failed to throw any light upon the case. All the sinuses illuminated clear, due to the thinness of the facial bones and skull. The radiogram showed frontals to be rudimentary and the right ethmoid sinus cloudy. The left ethmoids were cystic. Operation: A bilateral exenteration of the ethmoids and sphenoidotomy. A submucous resection of the nasal septum was necessary to obtain working space on the left side. The ethmoid cells were found to be cystic and the right cells were filled with granulations. Convalescence was uneventful, and the case made a happy recovery with a lessening of all the symptoms save for the tenderness of scalp.

Case 9.—S. D., age 68. Chief complaints were a persistent productive morning cough and a postnasal drip during the day which required constant clearing of the throat. Past history of a chronic nasal infection and bronchitis during the winter months. Examination showed the nasal membrane to be congested and the middle turbinates displaced by hypertrophied

ethmoid cells. Radiogram showed all sinuses to be cloudy and the sphenoid sinuses of average size. Operation: A bilateral exenteration of the ethmoid cells and sphenoidotomy. The right sphenoid remained well opened, but there was a tendency for the left sphenoid sinus to close by reformation of the anterior wall. The cough improved, but with each change of the weather there is a return of the coughing on rising, but it produces a clear mucus and not the former purulent discharge.

Case 10.—G. G., age 12. Chief complaints were an acute daily elevation of temperature to 100° with a productive cough. Past history: Just one year ago seen with a similar condition, and as there was a reformation of the tonsils and the adenoids, a second tonsil and adenoid operation was performed. For two weeks after the operation she had a dry irritable cough, which gradually improved. One acute infection of the sinuses with a cough during the winter. Examination: Lungs showed signs of enlarged glands and the radiogram was suggestive of mediastinal pathology. There was a postnasal discharge, and the larynx was very red. Treatment by the use of suctions and the nasal douche did not materially aid, so the child was sent to Colorado for the summer. As soon as the postnasal discharge cleared the cough stopped and the chest signs disappeared.

Case 11.—Mrs. W., age 30. Chief complaint, acute suppurative maxillary sinusitis of dental origin. Duration of one year with inferior dental treatment. At operation the ethmoid cells were found to be degenerated and filled with pus, and a subsequent exenteration of the cells was necessary to cure the antral infection. The ethmoid cells of the opposite side were not involved. This is an example of involvement by extension.

Case 13.—Mrs. B. C., age 64. Chief complaints were a cough from a postnasal drip with general muscular pains, more pronounced in the posterior cervical region. Examination: Nasal membrane coated with a thin purulent discharge, and the turbinates were atrophic. Operation: A double ethmoid exenteration and sphenoidotomy with no postoperative packing. Convalescence was uneventful until the fifth day, when a secondary hemorrhage developed from the right sphenoid

region, which required a postnasal plug before it was controlled. Packing removed on the eighth day, with no difficulty, though the discharge was offensive. On the tenth day another sharp hemorrhage developed, which required anterior and posterior packing. The next day the anterior packing was removed and the following day the postnasal plug was removed and following in its wake was a tablespoonful of foul discharge. No further packing was necessary and convalescence was natural. This case was the most marked secondary hemorrhage encountered and exemplified the necessity of a postnasal plug when a sharp hemorrhage is from the region of the sphenoid.

Case 14.—J. F. H. Chief complaints were coughing, postnasal discharge, and a history of an atrophic rhinitis for twenty years. Examination showed a typical atrophic rhinitis with large foul scabs and small turbinates. Extensive treatment failed to accomplish anything save temporary relief. The radiogram showed the ethmoid cells to be better ventilated than the frontals, and the anterior posterior picture showed the sphenoid sinuses to be unequally developed, the left being the larger. The frontals were small. Operation: Bilateral exenteration of ethmoids and sphenoidotomy. There was free bleeding from the right sphenopalatine artery which required packing. Several hours after operation there developed a hematoma of the right orbit from the hemorrhage, continuing and making its exit through the sphenopalatine foramen. Convalescence was uneventful save that the right sphenoid sinus was closed when seen after an interval of a month. An opening into the sinus revealed it to be very shallow, but a radiogram was made with a probe in each sinus, and it showed that they were open. The left sinus was filled with Beck's bismuth paste, X-rayed to ascertain its size, and it was found that the left sinus was very large and that the right sinus was small, with the sphenoid partition deviating to the right. After all the granulations had healed the membrane was moist and healthy and there were no more scabs. The irritation of the larynx is less and there is no further coughing except when exposed to an irritating atmosphere.

Case 15.—W. F. M., age 24. Seen in consultation to determine if the left sphenoid sinus was open. There had been

a curettement of the left ethmoid cells and what was thought to be the left sphenoid had been entered. The opening was situated high and posteriorly, but too far externally for the sphenoid sinus. An attempt was made to puncture into the sphenoid sinus, but the anterior wall was resistent and what appeared to be the anterior wall measured 8.5 cm. from the tip of the nose. A probe was placed in the cells that was suspected of being the sphenoid sinus and X-rayed and, as the picture showed, it was an ethmoid cell situated above and external to the sphenoid, and an entrance into the cranial fossæ could have been easily accomplished by the use of undue force or persistency.

Case 16.—W. A. L. Chief complaint was a progressive loss of vision to 1/200 in left eye, with central blind spot. Narrowed blurred field. Fig. 1. Acute pain about left eye for the past three weeks. Physical examination was negative, Wassermann negative. The ethmoid cells of the left side were hypertrophied, displacing and jamming middle turbinate against septum. The septum deflected to the right. X-ray revealed a large cyst of left ethmoid. Right ethmoids showed increased density. Septum to the right. Sphenoids of average size and slightly cloudy. Left middle turbinate upon removal had become flattened by atrophy. The bony septum was very thin from pressure. Upon puncturing the bulla the cyst was entered. Exenteration was simple. Anterior walls of the sphenoid removed. The ethmoids had undergone paperization and extended backwards and inwards until they completely obscured the sphenoid. No discharge present. Uneventful convalescence. Vision twelve days postoperative: Right 20/20, left 2-1/200. Four weeks postoperative, vision 20/20 both eyes. Fields and tension normal. Fig. 2. X-ray shows septum in midline and left ethmoids absent. (In order to demonstrate a cyst of the ethmoid its walls must be intact.) Case 17, Mrs. C., demonstrates the inability to show a cystic cavity after part of its wall has been removed.

Case 17.—Mrs. H. C. Chief complaints were progressive obstruction to nasal ventilation and drainage of one year's duration, with acute pain about eyes for past two weeks. There was an exophthalmus of the left eye. Examination: Nares completely filled with polyp and a profuse purulent dis-

charge. The left eye was displaced downwards and outwards with exophthalmus of 6 mm. Vision, 20/20 in both eyes, with normal fields. The general aspect of the left orbit presented signs of subacute orbital cellulitis. Upon palpation a cystic enlargement could be made out from the floor of frontal sinus at its inner angle. No tenderness was present. Exenteration of the polyps and degenerated ethmoid cells and drainage of sphenoid sinus was done at one sitting. Convalescence was protracted due to drying of the purulent discharge with the production of scabs. X-ray six weeks after operating failed to outline the tumor mass in the left orbital frontal wall. This was due to a partial removal of the wall of the cyst. The exophthalmus persisted, but the vision remained 20/20 in both eyes before and after operation. The discharge grew less and localized to the left naris in its anterior aspect. One day while making pressure over the tumor mass a stream of the purulent discharge was visible coming down from in the region of the frontal duct. This decided the issue that our enlargement along the floor of the frontal sinus was a purulent collection and connected with the frontal sinus or a supraorbital ethmoid cell. A radical operation was advised, but as there was no further pain and the discharge was minimum she decided to wait. This case illustrates how extensive and prolonged pathology may be in an orbit without affecting the vision.

Case 18.—Mrs. F. A. P., age 54. Chief complaints were obstructed nasal breathing and profuse purulent nasal discharge. Examination showed both nares filled by polypoid tissue and same coated with a purulent discharge. Operation: The polyps were removed and the right ethmoid cells exenterated and the right sphenoid sinus drained. The septum was found to be deflected to the left after the removal of some of the polyps from the left naris, and a resection of the septum was necessary before the left ethmoid cells could be exenterated. The tissues were very vascular and did not readily accept cocainization. Convalescence was marked by a rapid reformation of a vascular growth in the posterior ethmoid region, and one month after the first operation this new formed tissue was removed thoroughly and the pathologic report was that it was of an inflammatory nature. One year later patient returned with glowing reports of a healthy winter passed in

Texas and full of appreciation for having spent a year free from sickness. Examination showed reformation in the same region, left posterior ethmoid, and so a third operation was recommended. The reformed tissue was removed and a few remaining posterior cells were exenterated from above and to the side of the sphenoid. The sphenoid opening was enlarged and the partition between the sphenoid and ethmoid cells completely removed. The first two operations required nasal packing for persistent bleeding, but no packing was necessary for the last operation, and her convalescence was uneventful. The tissue which reformed before the second operation had the appearance of malignancy, but the laboratory report of chronic inflammation was sustained by the progress of the case.

Case 19.—Mrs. S., age 72. Chief complaints were a boring pain through the upper jaw, a purulent nasal discharge, and difficulty in mastication. Examination showed the right superior maxillary region to be swollen and the hard palate to bulge on the right side. The right middle turbinate was displaced by a granular mass, which bled profusely when a section was punched out for examination. The laboratory reported the condition as sarcoma, and no operative interference was instituted and the old lady died shortly afterwards. This was a case of malignancy of the ethmoid cells and the maxillary sinus.

Case 20.—Mrs. L. M. D., age 36. Chief complaints were cellulitis of the left orbital tissue and a foul purulent nasal discharge. Past history of severe headaches and pain about the left eye for the past year. Examination showed a fistula below the inner canthus of the left eye with swelling and redness of the orbital tissues. The left nares was filled with a foul caseous material. Under ether an external opening was made into the ethmoids by enlarging the present opening, the cells cleaned out as well as possible and a through and through drainage established. Strenuous antisyphilitic treatment was instituted and the parts irrigated with Dakin's solution. The acute symptoms subsided and the external wound healed with a very small scar and no deformity, but the internal destruction continued until there was a perforation of

the hard palate and the vomer came away en masse. This case was a gumma occupying the ethmoid cells.

Case 21.—E. A. M., age 3. Chief complaints were nasal discharge and large green foul scabs. The child was anemic and quiet and did not desire to enter into play with other children. Examination showed the middle turbinate to be coated with scabs, and a postnasal discharge filled the nasopharynx. The condition was treated by the usual routine suction and nasal douching for one year with no marked improvement. Then came the interval of two years of war. During this period the home treatment had been continued, but acute colds were numerous and scabs would form upon the cessation of douching. The radiogram showed the ethmoids to be of average size for a child of six years, but cloudy and there was an increased density over the antra. Operation: The anterior halves of the middle turbinates were amputated and the ethmoid cells curetted. At the same time the tonsils and adenoids were removed. Convalescence was slow, but the nasal discharge grew less and the scabs became less frequent. During the following winter there were less acute nasal infections and the child improved.

Case 22.—E. J., Jr., age 8. Chief complaints were recurrent acute infection of upper respiratory tract and a low grade fever for which no cause could be found. The tonsils and adenoids were removed in 1918 by an associate, and the record shows a satisfactory operation, but due to persistent obstruction to free nasal breathing a second operation was advised and performed by another surgeon the following year. No lasting improvement followed the second operation. Examination: The nasal membranes were dirty and coated with a thick mucopurulent discharge, the middle turbinates were of average size and there was a ridge and spur of the septum to the right. The nasopharynx was filled with the same discharge and there was a marked hypertrophy of the pharyngeal lymphoid tissue, especially opposite the soft palate. Suction treatment and nasal douching were advised and some improvement was noted. Operation was considered, but patient passed out of our care and remains a case of chronic ethmoid suppuration, which is causing his temperature and an obstruction to free nasal ventilation. Evidently the ethmoids were the

cause of the symptoms for which the unnecessary second operation was performed.

Case 23.—Mrs. W. A. W., age 36. Chief complaints were persistent headaches and postnasal discharge. Past history of several nasal operations, including a submucous resection of the nasal septum and amputation of the right middle turbinate. Examination showed the right ethmoid to be hypertrophied and displacing the stump of the middle turbinate towards the center. The left ethmoids were obscured by the middle turbinate. Operation: Right ethmoids exenterated and sphenoid sinus drained. The left middle turbinate was amputated and the ethmoid cells exenterated, and the sphenoid sinus drained. Convalescence was delayed by a reformation of the sphenoid walls, the right sinus being completely filled. This was removed twice surgically and once with cautery, with no avail. A history of a tumor formation in the scar of her abdominal operation which required radiation suggested the use of radium. A 25 mg. needle was inserted into the sphenoid sinus. Two hours after the initial exposure of thirty minutes her headache was better, so the following radium treatment was administered to the right sphenoid sinus:

Oct. 30, 1920. 25 mg. for 30 mins., brass and rubber screening.

Nov. 8, 1920. 25 mg. for 30 mins., brass and rubber screening.

The following day the opening was large enough to irrigate the sinus.

Nov. 24, 1920. 50 mg. for 30 minutes with same screening.
Dec. 20, 1920. 50 mg. for 30 minutes with same screening.
Dec. 31, 1920. 50 mg. for 30 minutes with same screening.
Jan. 10, 1921. 50 mg. for 30 minutes with same screening.
Jan. 27, 1921. 50 mg. for 30 minutes with same screening.
Feb. 9, 1921. 50 mg for 30 minutes with same screening.
Feb. 18, 1921. 50 mg. for 30 minutes with same screening.

Reaction in tissue and right sphenoid sinus deepening. March 21, 1921, two 12½ mg. needles inserted in the right sphenoid sinus for 40 minutes. April 4, 1921, the above needles for one hour into the same sinus. The left sphenoid cavity is larger than its fellow and the partition is deflected

to the right. The sinuses were radiographed with a zinc stearate solution acting as a filler, and the left is considerably larger. The radium would cure up the headache, and finally the right sphenoid became normal in size and shape and is now draining. Radiogram to illustrate measures necessary to ascertain if both sinuses were open.

Case 24.—Dr. A. M. L., age 44. Chief complaints were a chronic nasal discharge and headache. Past history of chronic maxillary and frontal sinusitis, for which a Caldwell-Luc antrum operation and several ethmoid exenterations had been performed. Examination showed a well drained maxillary sinus but a blocked frontal sinus on the same side (left). The remaining ethmoid cells were sclerotic and at operation the opening of the frontal duct could not be accomplished to a satisfactory degree. A second attempt several weeks later by removal of the cells in the uncinate process did not improve the drainage. Radium was tried by using the needles (25 mg. for two hours), encased in wax and retained in close approximation to the anterior ethmoid cells. Two weeks later patient returned with a glowing account of his freedom from headache and the lessening of the purulent discharge. A second exposure of 50 mg. for one hour with the same screening was administered, and upon the next visit to the office, three weeks later, the membrane was cleaner and more vascular and his condition greatly improved.

Case 25.—S. J., age 19. Chief complaints were persistent nasal obstruction and postnasal discharge after several nasal operations, including a submucous resection of septum and a thorough ethmoid exenteration. Three subsequent attempts at removal of the reforming excessive granulations and polyps were of no permanent avail. Seen in consultation with my associate, and advised radium treatment. Examination at this time showed the nasal membrane to be edematous and the ethmoid region refilled with polypoid tissue. As much as possible of this new formed tissue was removed, and three exposures of 50 mg. radium for two hour intervals were tried with only slight improvement. The question of protein sensitization was considered but not isolated. At one of the operations the orbit became filled with a hematoma, which displaced the eye outwards and forwards with no bad result. This was caused

by a piece of the lamina papyracea coming away in the biting forceps.

Case 26.—W. P. J., age 60. Chief complaints were a progressive obstruction to nasal breathing until complete obstruction had developed. Examination: Nares filled with polypoid tissue coated with purulent discharge. Operation: Polyps removed and degenerated ethmoid tissue cleaned out. Both sphenoid sinuses drained. Convalescence was marked by profuse discharge and the formation of a thick false membrane, which resisted removal by irrigation, and required separation before it could be removed. These radium exposures of 25 mg. in each naris for one hour and a half with needles cleaned up the tissue and reestablished ventilation. Before each radium treatment the false membrane was removed and the excessive granulations cleaned away with biting forceps.

Case 27.—J. L., age 21. Chief complaints were a lack of concentration, persistent ethmoid infection, causing a cough and elevation of temperature. Examination: Nasal membrane thin and red. Left middle turbinate cystic. Septum, spur to left. Suction was productive of a purulent discharge from ethmoid region. Operation: Submucous resection of nasal septum, amputation of cystic left middle turbinate and exenteration of cystic left ethmoid cells filled with pus. Uneventful until sixth day. Postnasal hemorrhage requiring packing of left sphenoid sinus. Later examination showed cessation of purulent discharge with marked improvement of patient's health and a return of a quicker apprehension.

Case 28.—T. L. W., age 22. Chief complaints were a lack of concentration, due to constant frontal headache and ocular pain. Both middle turbinates displaced centrally by hypertrophied ethmoid cells. Previous submucous resection incomplete, leaving a thickened superior half of septum. X-ray of frontal sinuses small and cloudy, ethmoids clear (in fact, too clear), sphenoid of average size. Operation: Submucous resection completed and exenteration of ethmoids and a double sphenoidotomy. Subsequently sphenoids reformed their anterior walls, which required a second and more complete resection. Convalescence was protracted but satisfactory, and as the drainage and ventilation became established headaches

were less annoying and concentration more astute, which allowed the patient to continue the study of medicine.

Case 29.—Mrs. O. M. D., age 54. Chief complaints were occipital headaches, postnasal discharge, obstructed nasal ventilation and progressive deafness. Past history of a chronic nasal discharge and a dermatitis of the external auditory canals. Examination showed the ethmoid cells to be hypertrophied, displacing the middle turbinates towards the median line. Operation: A bilateral exenteration of the ethmoid cells and sphenoidotomy. The septum was so thickened above as to obstruct the removal of the high cells, so a submucous resection of it was necessary before the ethmoids could be exenterated. There was a profuse mucopurulent nasal discharge for the first week, which subsided and, much to our surprise, the dermatitis of the external auditory canals improved and there was also better ventilation of the eustachian tubes.

Case 30.—O. P., age 22. Chief complaints were a persistent discharge from right ear, for which a Heath semiradical mastoidectomy was performed fifteen years ago. An atrophic rhinitis with scabs complicated the aural condition, and was thought to be a prominent factor in the continuance of the discharge. A marked septal deformity to the left interfered with any surgical procedure, so a submucous resection of the nasal septum was first done. The ethmoids were found to be degenerated and were exenterated. The anterior wall of the sphenoid was removed. Convalescence was uneventful until the third day, when a hematoma of the septum was apparent. This became infected and required drainage by a tube. The nasal pathology improved and the purulent discharge and scabs abated and with their departure the aural discharge ceased. The antral cavity is easily inspected and well drained. The membranum tympanum has a large perforation in its inferior third, with limiting adhesions which divide the middle ear into compartments, the lower being a direct drain of the eustachian tube. This case shows the possibility of an aural discharge being caused by suppurative ethmoiditis.

Case 31.—Mrs. T. A., age 35. Chief complaints were rapid loss of vision and an acute pansinusitis. In one week vision was reduced to O. D. 10/200, O. S. blind. There was a marked papilledema with swelling of 3 D. of the nerve of the head and

a few small hemorrhages about the disc. Fields as shown (Dr. E. C. Ellett). Examination showed an acute suppurative left pansinusitis. A radiogram revealed the left frontal, ethmoid and antrum to be filled with granulations or pus and the right sinuses to be cloudy but not filled. The sphenoid sinuses were cloudy. Operation: Sphenoidotomy and drainage of both ethmoids. The left antrum was thoroughly drained by a Canfield operation. The pus from the sinuses showed a mixed infection of streptococci and staphylococci. In ten days she counted fingers with the former blind eye. On the tenth postoperative day her vision was 20/30 in both eyes, and before the end of the month 20/20 J. 1 in both eyes. Case was formerly reported by Dr. E. C. Ellett.

Case 32.—Mr. E. P., age 52. Seen in consultation on the fourth postoperative day. Past history: Recurrent infection of the ethmoid sinuses and a history of an old luetic infection. Present history: Four days ago a bilateral exenteration of the ethmoids, after the technic of Mosher, which had progressed normally until this morning, when a headache began about nine o'clock. Morphin, gr. 1/4, was administered for the pain, and patient went into a stupor. Examination, 4 p. m., of the same day (fourth postoperative) did not show anything different from the ordinary postoperative nose of the same period. The general appearance of the patient was that of pneumonia, as there was a cyanosis with Cheyne-Stokes breathing. Sensorium was very cloudy. There was no rigidity of the neck and no Babinski reflex or Kernig's sign. Spinal fluid was withdrawn and was cloudy and under pressure. This showed on smear and culture a pneumococcus. The blood also showed a pneumococcus after twenty-four hour growth. The patient did not arouse and died on the night of the fifth day. Before death the lungs showed the first stages of a pneumonia. This was an example of where the infection extended up into the cranial cavity by way of the lymphatics or blood stream.

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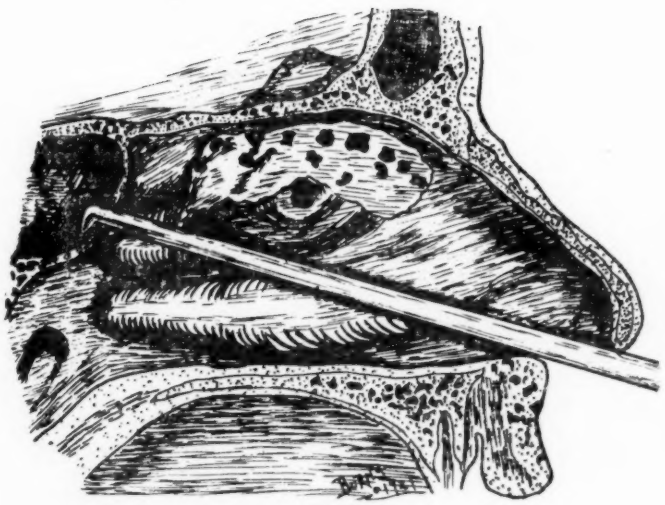

Fig. 1

Showing the relationship of the fleshy posterior tag of the middle turbinate and the sphenoidal sinus.

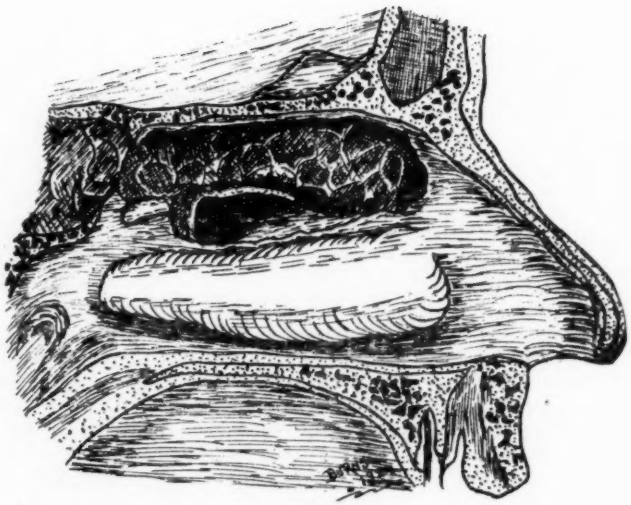

Fig. 2

The complete extenteration and an enlargement of the ostium into the antrum.

XLVII.

THE PRESENT STATUS OF SKIAGRAPHIC INTERPRETATION AS AN ADJUNCT IN THE DIAGNOSIS OF CATARRHAL AFFECTIONS OF THE ACCESSORY SINUSES.*

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I think most of us have long since, as a routine procedure, sent our obscure sinus cases to the roentgenologist for his advice and have largely been guided by his report in our subsequent treatment of that particular case. If, for example, exudate was said to be present in the anterior ethmoid cells of a given side these cells were immediately opened, even though direct examination had revealed nothing abnormal nor had the symptoms pointed particularly to those individual structures. How often at the time of operation have we been disappointed to note the absence of all pathologic changes, and it required a considerable stretch of the imagination to convince ourselves that the operation had really been indicated, much less that the benefits which might accrue would bring about a complete or even partial relief of the symptoms. That we have been placing too great confidence in the X-ray reports, to the detriment of our own better judgment, has slowly been dawning on some of our minds for several years. Levy[1] in an article on diagnosis of sinus disease states that the X-ray is overestimated. LaRue in a discussion of this paper says operation often shows positive disease while the X-rays even negative or even vice versa. Ballenger,[2] in an exhaustive study, says a secondary place should be given to the X-ray and the main reliance should be placed on clinical means.

Boot,[3] while discussing this subject, said many of his reports had been absolutely wrong. Mithoeffer[4] says he considers the X-ray picture as an adjunct of secondary importance.

*Read before the American Laryngological, Rhinological and Otological Society, 1922.

Culp[5] cites a case in which a prominent roentgenologist, after taking numerous negatives, stated no trouble existed in the sinuses. Shortly afterward she was operated upon and both antra were found filled with cheesy matter and polypoid tissue degeneration. Chase[6] states it is possible to have pus present with practically no shadow on the plate. Guttman,[7] on the other hand, had a case which showed marked cloudiness of the antrum, which on being observed was found to be healthy. Dean[8] writes that radiography is very unreliable in infants and young children. Not so clear as in adults. The sphenoid and posterior ethmoids being almost always blurred on the plates. Long says the X-ray is of considerable service, etc. All these opinions have been expressed within the past twenty-four months. It is my purpose to take these statements as a text for this paper and endeavor to present a frank and unprejudiced discussion of the actual value of X-ray plates to us as rhinologists as an aid to the diagnosis of inflammatory conditions within the sinus cavities.

Let us first briefly consider the standard methods of exposure which are instantly recognized, omitting those which present curious and bizarre shadows making the anatomic orientation difficult for the average laryngologist. The frontal sinus (Caldwell):

1. Posteroanterior. For size and shape; shadow.
2. Lateral. For depth and thickness of anterior wall.

The maxillary. Posteroanterior for shadow and size.

2. Lateral. For relation to teeth.
3. Extended chin (Waters and Waldron). Shadow for comparison with opposite side.

The ethmoid:

1. Posteroanterior. For shadow and extent.
2. Oblique (Rhese).
3. Stenoscopic. For anatomic configuration.

The sphenoid:

1. Lateral. For shadow as compared with ethmoid. Size and shape.
2. Posteroanterior. Shadow.
3. Extended chin. Shadow and size.
4. From above, film in mouth (Pfahler). Shadow, shape and size.

These I think include the methods that today are in general use, the plates from which we are accustomed to examine and confirm the report of the roentgenologist. If we were asked which sinuses in their order were shadows more easily distinguished we would probably say: (1) Maxillary, (2) frontal, (3) sphenoid, and (4) ethmoid. This order would perhaps also check up with our clinical findings as far as confirmation was concerned. Why then do we have such divergent expressions of opinion as to the actual value of radiographs for diagnostic purposes, and why have so many of us been disappointed after confirming our treatment to the X-ray disclosures? Sauer says that improper technic and faulty interpretations are usually the basis of its lack of efficiency, and positive findings frequently depend upon the overzealousness of the interpreter rather than upon the pathologic process. It is said the X-ray never lies. It shows what is there if one can judge by the superadjacent tissues. This, to my mind, is the crux of the entire situation. The shadows must be properly interpreted in order to be of value and, as the personal equation must enter largely into the readings, it is essential that the reader must have checked his experiences with the subsequent surgery, so that his statements as to findings will be reliable data for the rhinologist to unhesitatingly follow. This will undoubtedly occur when the rhinologist himself becomes an expert interpreter of roentgenograms. Before considering the individual sinuses let us for a moment diverge and discuss some of the common causes of errors in interpretation.

1. Faulty Position.—All of us at one time or another have seen ordinary photographs which could hardly be recognized by even a member of the family. Why cannot the same be true of an X-ray photograph? As a matter of fact, it may distort to such an extent that one receives a wrong impression of the plate with a consequently false interpretation. This can be laid to an error in technic and not the reading, but would hardly occur in a well grouped series of exposures.

The presence of air in the sinuses appears to exert considerable influence upon the shadow as to sharpness of outline. Secretion in the sinuses, even thick inspissated, would appear to play a rather unimportant role, although occasionally

one could observe a line of demarcation at the fluid level. The shadow, as a rule, however, is not greatly influenced. Swollen and inflamed mucosa gives a denser shadow than exudate and has often been confused with it.

Its relative value in the individual sinuses.

Maxillary.—It is here that the X-ray as a diagnostic measure has its greatest value in sinuology. By means of the anteroposterior exposure comparison with the opposite side is easily and readily made and constitutes the basis of the interpretation, which does not apply so well to any of the other sinuses, not excepting the frontals. Even here, however, the possibilities of an error cannot be overlooked, which may be caused by a shadow due to asymmetry of the bones of the face or the thickening of the lining mucosa in one of the bones of the face or the thickening of the lining mucosa in one of the antra, the result of a former infection.

Frontal.—Much information can be obtained from good skiagraphic plates, provided the proper lines of exposure are observed. The posteroanterior for all around purposes is probably the best, as it not only gives the shape, extent and position of the sinus, but is very liable to give a fairly accurate reading of the condition of the sinus cavity. While shadows were occasionally observed in which no clinical evidence of inflammation was present, on the whole the plates furnished data which could be relied upon and, coupled with the clinical symptoms, were of considerable value in the subsequent treatment of the affection. The lateral position gives information as far as the anatomic configuration of the sinus is concerned, such as its depth, the thickness and position of the anterior and posterior walls, but throws little light on the contents or condition of the mucosa. Skiagraphs of the frontal sinus can then be considered of such value as to warrant them in all cases before operative procedures are applied.

Sphenoid.—Opinions of both the sinuologist and roentgenologist seem to be converging, in that, while these sinuses are distinctly within the scope of the X-ray, it applies more particularly to the anatomic configuration and relations to surrounding parts and less to the actual pathologic condition which obtains within the sinus cavities. The continual ex-

perimentation with new positions would seem to lend weight to this assertion.

Cushway,[9] for example, has tried all the various positions recommended and has been extensively experimenting in this direction, but thus far the results had been disappointing. Long,[10] in a comparatively recent writing, considers it of considerable service in demonstrating the size and shape of the sphenoid sinuses, but of limited assistance in giving information as to the gross pathology. As far as the positions of exposure are concerned, there is little to choose between them, as long as there is no anatomic distortion which makes orientation difficult. The anteroposterior position, bringing the sphenoid out through the orbital cavities, seems to have the greatest vogue, generally speaking, but the oblique positions of Rhese and that of Pfeiffer enjoy no little popularity, particularly in the larger cities of the East. None of these methods has proven satisfactory in my own work. I have never been certain that the operation would disclose the precise findings one had been led to expect and have usually not been disappointed. Many times exudate has been positively diagnosed, of which on operation no signs were visible, and vice versa. I have long since discarded the roentgenogram as anything more than a possible confirmatory means to a diagnostic end. Bowen,[11] a roentgenologist, has come to the conclusion that neither the Pfeiffer position nor the reverse one is of much value in the diagnosis of sphenoid sinus empyema. Let us hope the method recently devised by Pfahler,[12] with the film held tightly against the soft palate and the rays directed downward, may prove more reliable.

Ethmoid Labyrinth.—It is here in affections of these cells where most help is needed from the skiagraph that we get the least. When I first had the opportunity to study skiagraphic stenoptican views of the ethmoid, I felt that the X-ray had disclosed its hidden pathology and that hereafter it would be an open book. Subsequent experience has only added to my disappointment, which seems to be shared by many of my colleagues. Thus, Wherry[13] says the field of usefulness of the ray, as applied to the ethmoid heretofore, has been most unsatisfactory. The reason for this is that a shadow or haziness in the ethmoid region does not always indicate an exudate

or any change in the cells, as an hypertrophied middle turbinate will give this appearance on the film. Purulent secretion in one or more of the cells will usually not influence the density of the shadow, and even a thickening of the lining mucosa will scarcely affect it. The most reliable indication, from a roentgenographic standpoint, for a pathologic process is the blurring of the intercellular septa. If these cellular walls do not come out clear and sharp in several exposures, undoubtedly a breaking down or a reabsorption has occurred, which may be due to either a hyperplastic or suppurative process. If the absorptive process has caused the formation of one large cavity, the reading will of course be plain, but we are not considering the simple cases, but rather those that are more commonly met with. Summing up, then, the comparative efficiency in the various sinuses, let us arbitrarily place the ratio of value about as follows: Maxillary sinus, 85 per cent efficient; frontal sinus, 75 per cent efficient; sphenoid sinus, 40 per cent efficient; ethmoid labyrinth, 25 per cent efficient.

This does not mean that in a given number of cases this percentage will be found to be absolutely correct, but rather the relative value of assistance the ray gives in disclosing the pathologic condition present in the individual sinuses.

In conclusion, one must frankly state that the mere presence of a shadow in a sinus cannot be considered a reliable indication for operative intervention unless accompanied by the usual clinical symptoms pointing to disease in that particular sinus.

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XLVIII.

OTITIC PYEMIA WITHOUT SINUS THROMBOSIS.

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In 1885 Schwartze[1] called attention to otitic pyemia without sinus thrombosis, which he thought was caused by an osteophlebitis of the mastoid bone. Nothing further appeared in the literature on this condition until Körner[2] published the results of his studies seventeen years later. Although there may be some question relative to the possibility of the production of an otitic pyemia by an osteophlebitis, there can be no question of the value of Körner's deductions of this subject.

He pointed out that this type of pyemia occurred chiefly in children and young adults. It is usually a complication of an acute otitis media or mastoiditis. The metastases were usually found in the joints, serous cavities and muscles; rarely in the lung.

He explained the reason for the infrequency of lung emboli, in that the bacteria are not included in thrombus particles, therefore passing through the lung and, as Wyssokowitsch[23] has shown, lodge in the capillaries of organs in which the circulation is slow—i. e., the spleen, liver, kidneys, bone marrow, etc.

Körner compiled the following statistics, based on 51 cases, many of which were selected from Hesler's[22] compilation on otitic pyemia.

The ages represented were as follows:

14 cases ranging from 0-10 years.
16 cases ranging from 11-20 years.
14 cases ranging from 21-30 years.
5 cases over 30 years.
2 cases, the age not given.

The outcome of these cases was tabulated as follows:

14 were cured by a paracentesis or no treatment.
17 were cured by a simple mastoid operation.

2 by a simple mastoid, plus laying bare the sinus.

5 cases required a ligation of the jugular.

The mortality was 25.5 per cent, or 13 cases. The causes of death were given as follows:

In eleven cases there was a phlebitis of sinus or vein.

In one case there was no infection of the vein or sinus.

In one case the cause was not given.

The prognosis is therefore more favorable than in true sinus thrombosis.

In addition to the production of this type of pyemia by an osteophlebitis, it has been shown that the same condition may be brought about by bacteria passing directly through the sinus walls, or even through the veins and lymphatics of the middle ear.

The above statements have been amply borne out by the case reports of Uffenorde,[3] Breiger,[4] Urbantschitsch,[5] Brühl,[6] Wyssokowitsch,[7] Politzer,[8] Mueller,[9] Rimini,[10] Heine,[11] Schwabach,[12] Zufal,[13] Gruenert,[14] Kobark,[15] Heymann,[16] Massier,[17] Luc,[18] Huyer,[19] Zaalberg,[20] Kopetsky,[21] Hessler,[22] Wyssokowitsch,[23] Kopetsky,[24] Shambaugh[25].

Bacon,[26] Page,[27] McKinney,[28] Boot,[29] Phillips[30] and Oppenheimer[31] have reported cases which agree with the clinical picture described by Körner, but no mention of this is made by the authors.

The two cases reported by Dr. Page are rather characteristic:

Case 1.—A child, two years of age, had a double otitis media following influenza. During her illness her temperature would rise as high as 104 to 106 degrees F., daily. Dr. Page performed a double mastoid operation, but found little destruction of the mastoid cells. Both sinus walls had a dirty grayish appearance, especially the left, but no thickening or other signs of thrombosis were present. He did not open the sinuses. The temperature came down gradually. Eight days later the gauze drainage was removed on the left side; this allowed the soft parts to lie against the vein for twenty-four hours. During this time her temperature came up to 105+. After separating the soft parts from the vein wall and wiping the wound with 5% dichloramin T, the temperature became normal.

Case 2.—A boy of eight years of age was sent home from the hospital on the tenth day following a mastoid operation. In this case the sinus had also been exposed. Wound repair was slow and the sinus had not yet been covered with granulations. He suddenly developed a temperature of 105 F., and was sent back to the hospital. His wound was cleaned up and rubbed with a dichloramin T solution, following which his temperature returned to normal the next day, his white count remaining high for several days.

Some of the conclusions drawn by Heymann[16] are of importance in the development of a paper on this subject. His results after infecting the superior longitudinal sinus of twenty dogs and the lateral sinus of six monkeys by placing a tampon soaked in a culture of streptococci or staphylococci in contact with the sinus wall for varying lengths of time show that:

1. The mere application of bacteria or rubbing them into the sinus wall or even when the wall was scarified, plus the application of germs, did not cause the formation of a thrombus.

2. Pressure of a tampon on the sinus wall also produced no results.

3. When the tampon was saturated with bacteria, thrombosis did occur.

Therefore in a perisinus abscess containing virulent bacteria we have conditions favorable for the production of a thrombus.

4. It is not necessary to have a thrombus to produce an otitic pyemia. The germs may pass directly through the sinus wall. Heymann was able to demonstrate the durchwanderung of bacteria through the sinus wall to the inner side of the endothelium. This durchwanderung may also occur through the walls of the small veins of the middle ear. In several instances he found parietal thrombi which were not infected, but the adjoining wall showed germs penetrating through its inner layers.

Weigert also noticed the einwandurung of bacteria in the veins of the mastoid.[32]

The following case agrees, on the whole, with Körner's osteophlebitic pyemia:

Mrs. S., age twenty-seven, was referred to me by her family physician. She had been suffering with severe pain in the right

ear for about one week. Her drum had been incised and the ear was draining freely. She developed a severe pain in her mastoid process, with some edema and tenderness, two days before coming to me, and her doctor was obliged to use opiates to relieve her pain. The patient was undernourished. Examinations of her heart and lungs were negative. Her temperature on admission to the hospital was 101.2 F. Urine examination was negative. White blood count was 10,500. A smear from her ear showed a streptococcus. Examination of her ear showed an edema over the mastoid process, with considerable tenderness to pressure over the tip and antrum. There was considerable dropping of the posterior superior canal wall.

A simple mastoid operation was performed at once. The operation revealed considerable destruction of the mastoid cells. The sinus was not uncovered. Six days after her operation she developed a severe follicular tonsillitis and immediately following this an otitis media of her left ear. The drum was incised, but notwithstanding the fact that the drainage was ample and that every precaution was taken a mastoiditis developed on the left side. A smear showed that we were again dealing with a streptococcus infection.

Several X-ray pictures showed an increasing involvement of the mastoid cells. Again the patient suffered with violent pain, high temperature, edema and tenderness over the mastoid. The left mastoid was therefore opened two weeks after her first operation. Pathologically we were dealing with a hemorrhagic mastoiditis, streptococcus type.

There was not much destruction of bone, and the sinus was not laid bare. Her convalescence now progressed favorably until five weeks after her first and three weeks after her second operation, when on May 25th, I was called to her home, to which she had been discharged from the hospital. I found her in bed bathed in sweat. Her temperature was 106 F., this following a chill, which I was told lasted for twenty minutes.

She was readmitted to the hospital that evening. An internist was called. Erysipelas and tonsillitis were excluded. The lungs and heart were negative. Laboratory tests failed to show the presence of malaria plasmodia. The Widal test was

negative, as was the Wassermann. Her white blood count was 10,400. Urine examination showed only a trace of albumin. A blood culture was taken, which later also proved to be negative. The patient's general condition was below par. There was delayed healing of both mastoid wounds; both ears were dry. She had a severe frontal headache and complained of pain over her mastoids and neck, especially on her right side.

On the morning of May 26th, her temperature was 102.2 F., pulse 132, respiration 24. She had another chill twelve hours after being admitted to the hospital. The third chill followed twelve hours later.

About this time she developed an arthritis in her left ankle, which became quite red and swollen. The patient continued to have chills and sweats on May 27th, 28th, 29th and 30th. On May 29th another blood culture was made, which also was negative. A white blood count showed 9,800. This day she developed an arthritis in her left hip, the swelling and pain in the ankle having improved in the meantime.

On June 1st we decided to do an exploratory operation. There were no localizing signs or symptoms, excepting that the patient suffered more pain on the right side. This side was therefore opened first. Here we found an extension of the bone involvement posteriorly over a considerable area with partial destruction of the bone. The sinus was uncovered over a large extent. Its appearance was normal in every respect. The left mastoid was now reopened, but no pathologic conditions were found in the mastoid or in the appearance of the sinus. In view of the fact that it would be extremely hazardous to open both sinuses, we determined to puncture them and, if negative, culture the blood so obtained.

This was carried out after thoroughly sterilizing the mastoid wounds. Both sinuses bled very freely upon inserting the trocar. The bacteriologic examinations of both cultures was negative. The postoperative history is as follows:

The patient had no more metastasis after her operation. Her chills continued every twelve hours up to June 9th, excepting on June 6th, on which day she had but one chill in twenty-four hours. The arthritis cleared up in about ten days following the operation. Her regular temperature continued until June 13th.

CASE SUMMARY.

In a patient convalescing from a double simple mastoid operation, there suddenly developed chills, fever and sweats. Typhoid, malaria, erysipelas, tonsillitis, syphilis and the heart and lungs were ruled out as causative factors. The leucocyte count was never over 10,400, with the blood culture repeatedly negative. Under observations the patient developed an arthritis of the knee and hip of the left leg. She had continued severe headaches, especially on th right side, with tenderness on the same side. Fundus findings were negative.

Reopening mastoids disclosed an extension of the involvement of the right mastoid posteriorly. Both sinuses appeared normal and bled freely upon the insertion of a trocar. Cultures inoculated with this blood proved sterile.

CONCLUSIONS.

I feel that the above findings warrant a diagnosis of otitic pyemia without sinus thrombosis. The causative factor being located in the area of diseased bone posterior to that originally removed from the right mastoid; notwithstanding the fact that the differential diagnosis between pyemia with and pyemia without sinus thrombosis is at present extremely difficult. The main difficulty lies in the fact that it is very hard to rule out the presence of a parietal thrombus.

TREATMENT.

There are a sufficient number of cases of this type reported in the literature to warrant them some consideration relative to their treatment. What form of treatment should we adopt in a case presenting the symptoms of an otitic pyemia, particularly in a younger person, where the sinus shows no abnormality, and especially in the absence of a perisinus abscess? Should we not pause before proceeding with ligation and opening the sinus at once, even though there be a possibility of a thrombus? Would it not be advisable to thoroughly clean up the mastoid and lay bare the sinus, waiting at least forty-eight hours before proceeding with sinus ligation? If the sinus looks sufficiently suspicious one may use a trocar, even though the results may lead one astray where a parietal thrombus exists. If we proceed wth the radical operation unnecessarily

we have produced an unsightly scar. We may bring about an extension into the petrosal sinus. Death may supervene due to an insufficiency of the opposite vein. Neither are we certain that emboli may not continue to be thrown into the system through the circulation of the opposite side. If we are dealing with a bilateral affair under the same conditions there is even more reason for treating this condition with conservatism.

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XLIX.

REPORT OF A CASE OF THROMBOSIS OF THE SUPERIOR PETROSAL SINUS OF OTITIC ORIGIN, PRESENTING A SYMPTOM COMPLEX STRONGLY SUGGESTING THE DIAGNOSIS.*

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In considering the diseases of the sinuses of the dura mater secondary to otitis, one finds the literature relating to purulent inflammation of the sigmoid sinus rich in the reports of cases diagnosed and operated with curative results.

Zaufal[1] was probably the first to suggest the operative treatment of sinus thrombosis. With the addition of ligation of the jugular vein and the use of the jugular skin fistula, as practiced by Alexander, the percentage of recoveries is now about 75 per cent.

The symptoms of sigmoid sinus thrombosis are fairly well defined and generally the diagnosis is made without difficulty.

Unfortunately phlebitis of the other sinuses is not so easy of diagnosis. Possibly the lack of clear cut symptoms may account for the sparsity of the literature on primary petrosal sinus thrombosis. Quite a few cases of primary sigmoid sinus thrombosis with extension into the superior and inferior petrosal and even the cavernous sinus have been reported in the literature.

In those cases of extension into the petrosal sinuses the symptoms most frequently observed are swelling of the veins in the temporal region; thrombosis of the retinal veins (Politzer[2]); attacks of epistaxis and epileptiform seizures. When the process has invaded the cavernous sinus a clot will occasionally form in the ophthalmic vein, when there will occur engorgement of the vessels of the orbit, disturbances of vision,

*Read before the Philadelphia Laryngological Society, Feb. 7, 1922.

photophobia, paresis or paralysis of the motor oculi, abducens and trochlear nerves; exophthalmus, ptosis, edema of the eyelids, neuralgia of the trigeminus, and rarely, sloughing of the orbital tissues[2]. When this symptom complex occurs the diagnosis of thrombosis of the cavernous sinus may readily be made; however, at this stage operative measures promise but little.

Thrombosis of the superior petrosal sinus as a primary condition occurs, perhaps, oftener than has heretofore been suspected, but for lack of conclusive diagnostic symptoms it has remained unrecognized. Recently the opportunity was afforded of observing a case of primary superior petrosal sinus thrombosis with extensions posteriorly into the sigmoid sinus, and anteriorly into the cavernous sinus with fatal termination. The symptom complex presented by the case when first seen permitted of but one diagnosis. The case was twice operated, in spite of which the life was not saved. At autopsy the clinical diagnosis was confirmed. It is because of the above facts that the case is reported.

History (as related by patient).—Family history not obtainable in detail, but so far as could be ascertained it is negative as to the present condition.

Past History.—Patient states that he has always been in good health. Two years ago, following an abscess of the left ear, a chronic discharge resulted. At the same time there was an abscess on the back of the neck (apparently a carbuncle). The ear was poulticed and discharged. At first the discharge was odorless, but later became offensive. He has never had bleeding from the ear.

History of Present Illness.—Patient states that up until about three weeks ago he enjoyed good health, with the exception of the intermittent discharge from the left ear. At this time he developed a "cold on the left side of the face," with severe pain extending up over the region of the left temple, along the lower jaw and over the face. (In other words, that region supplied by the trifacial nerve.) The pain was very severe over the side of the head in front of the ear. Patient states that he does not have headache, nor has he had headache at any time during the past three weeks. He claims that at times he has felt chilly, developing into a feeling of warmth

and fever, followed by sweat. His answers are prompt and intelligent.

Examination.—The patient appears rather thin, considering the relatively short duration of his illness.

Nose.—Externally the nose is inclined to the right. The nasal septum is deviated high up to the left and low down to the right. There is a bilateral spine along the suture line. The left middle turbinate is visible before shrinking, but is in contact with the septum and with the lateral wall. The inferior edge of the left inferior turbinate is hyperplastic. The right middle turbinate is not visible before shrinking. The inferior edge of the left middle turbinate is hyperplastic.

Throat.—Secondary catarrhal pharyngitis. Tonsils medium sized and submerged; no deposit on pressure.

Functional hearing test revealed moderate reduction of hearing on the left side.

Otoscopic.—A. D. Membrane intact, rather dull, opaque; long process of the anvil not visible because of the opacity. Hammer handle slightly foreshortened. Short process of the hammer quite prominent. Very limited motion with the Siegel otoscope.

A. S.—The greater portion of the membrane has been destroyed. Granulations on the inner wall are visible through thick yellowish purulent secretion, which has an offensive odor characteristic of diseased bone.

Examination of Cranial Nerves.—I, II, III, IV, VI, VII, IX, X, XI, XII nerves on both sides appear and perform normally. The V nerve on the left side presents neuralgia according to the history, but is normally sensitive to all stimuli. The VIII acoustic branch is normal (the hearing impairment was due to the middle ear suppuration on the left side.) The vestibular branch is normal on both sides.

Kernig's sign negative; Babinski is negative; knee jerks perhaps a trifle exaggerated, but bilaterally equal. No ankle clonus.

Further Examination.—Patient has a spot of tenderness to deep percussion about 7 cm. posterior to a line perpendicular to the outer canthus of the left eye and about 7 cm. above a line drawn from the outer canthus of the left eye to the upper

attachment of the auricle. The spot itself is about 3½ cm. in diameter.

EYE FINDINGS.

External Examination.—Eyes move well and together in all directions, with nystagmic jerkings when looking to the extreme left and when extreme right. No nystagmus when looking straight ahead.

Pupils round, black, equal in size; react promptly to light, accommodation, convergence and consensually. Vision O. D. 6/6, O. S. 6/6.

Direct Ophthalmoscopic Examination.—O. D. Disc round, sharply defined with the exception of the nasal margin, which appears slightly fuzzy, but within normal limits. Physiologic cup medium size, well defined; lamina cribrosa visible; size and distribution of the vessels normal; fundus, including the macular region, normal.

Direct Ophthalmoscopic Examination.—O. S. Disc round; physiologic cup medium size, sharply defined; size and distribution of vessels normal; fundus, including the macular region, normal; no evidence of choking or inflammation of the disc.

Roentgenographic Findings.—Stereographic roentgenograms made of the head in both right and left lateral positions reveal a shadow on the left side beginning at the frontoparietal suture about midway between the vertex and base, posterior end about one inch wide and the anterior end about one-half inch wide. Irregular in shape and extending forward about 2 inches from the frontoparietal suture and is suggestive of abscess formation.*

Blood Count.—Hemoglobin, 80 per cent; erythrocytes, 4,500,000; leucocytes, 10,000.

Urine.—Amber; acid; sp. gr. 1025; clear; faint trace alb.; sugar, quantity neg.; diazo, acetone neg.; casts neg

Crystals, leucocytes, erythrocytes, bacteria, epith. cells, normal.

*Because of the misinterpretation of the roentgenograms, the name of the radiographer is withheld.

Spinal Fluid.—Clear, normal pressure; reaction alkaline; sp. gr. qs. not suf.; globulin neg. to ammonium sulph.; cell count 8 per cm.

In discussing the case with the patient's family physician, Dr. C. F. Hadley, of Camden, Dr. Mackenzie stated positively that the case was not one of brain abscess as was suggested by the X-ray specialist, besides, an exact diagnosis ot the case would need to satisfy the following history and findings:

1. An illness of comparatively sudden onset.
2. Agonizing pain over the region supplied by the trigeminal nerve without headache.
3. Fever characteristic of pyemia.
4. Emaciation and pallor (without icterus).
5. Discharge from the ear with the charactertistic odor of dead bone.
6. A blood count showing leucocytosis.

The diagnosis of primary thrombosis of the left superior petrosal sinus of otitic origin was made by Dr. Mackenzie as the only condition that could satisfy the totality of symptoms and findings enumerated above. Operation was considered imperative and was accordingly done.

The patient was operated the same day, November 27, 1921.

The usual postauricular incision through the skin, soft tissue and periosteum was made. The periosteum was elevated from the underlying bone down to and including the tip. The membranous external canal was separated from the superior, posterior and inferior walls of the osseous canal to the tympanic membrane. The mastoid process was opened with Alexander No. 10 chisel and was found to be generally eburnated. The antrum was opened and the bony ridge protecting the facial nerve was levelled to the floor of the canal. The lower osseous wall of the canal was levelled to the floor of the tympanum. The upper wall of the canal levelled to the tegmen tympani. The inner wall of the antrum was necrotic. This necrotic area was evident in the triangle bounded posteriorly by the anterior margin of the sigmoid sinus, superiorly by the tegmen antri and anteriorly by the external and superior semicircular canals. The necrotic area led toward the edge of the petrous bone where the superior and posterior surfaces meet—that is, the location of the superior petrosal sinus posterior to the

osseous labyrinth. The inner table of the mastoid overlying the sigmoid sinus was intact and to all appearances normal. The middle ear cavity was carefully curetted and a remnant of the hammer removed. The eustachian tube was curetted. The healthy bone overlying the sigmoid sinus was then removed and its surface appeared normal and glistening; however, following finger compression the sinus tended to refill with blood unusually slow, the sinus with its contents had a putty like resistance. It was then decided that there must be a thrombus in the sigmoid. The deep jugular was then ligated with two ligatures and divided between the ligatures. The sigmoid sinus was next incised, but without a flow of blood. The incision was enlarged and a red thrombus was uncovered. The thrombus was too large and the vis a tergo was insufficient to expel it. For this reason a horizontal incision was made through the scalp and periosteum extending from the posterior margin of the original external incision backward about two and a half inches. The underlying bone was bared and a groove in the bone was cut with the large Luer bone forceps for a corresponding distance. The lateral sinus was exposed half way to the torcular and the original incision of the sinus was extended posteriorly another inch and a half. A small spoon curette was introduced into the sinus posteriorly to the torcular when the thrombus was removed quite intact. Iodoform gauze pack was introduced into the wide open lateral sinus. Plastic after Panse. Iodoform gauze wicks were introduced into the wound cavity; outer plain gauze dressings and bandage were applied. The fact should be emphasized that there was no erosion of the inner cortex covering the sigmoid sinus. Neither was any cholesteatoma found.

The left superior petrosal sinus was not opened because the patient was in a very poor condition, and it was hoped that the expelling of the clot from the left sigmoid sinus had served to empty out the left superior petrosal sinus.

On the third day following operation (November 30, 1921,) the patient's temperature had fallen to 98 F., and remained below 98 F. until the evening of December 2nd, when it reached 99 F. The following morning it had fallen to 97.8 F., but reached 100 F. the evening of December 3rd. On the

morning of December 4th the first redressing was made, at which time the patient's temperature was 99 F. At 2 p. m. the same day the temperature was 98.2 F., but by 5 p. m. it had risen to 101 F. and at 10 p. m. it was 101.6 F. No chill was reported by the patient, nor was any noticed by the nurse. Aching of the teeth on the left side was complained of. There was no headache; no changes in the eye grounds observable at the bedside with the electric ophthalmoscope. Babinski was negative. There was no spontaneous nystagmus when looking straight ahead. The spinal fluid was normal. The blood Wassermann was negative, as was also the spinal fluid. The leucocyte count was 11,000.

December 7th, following the third redressing, the patient developed a chill with a rise of temperature to 103 F., followed by a fall to 99.6 F. The patient still complained of pain in the teeth on the left side. No fundus changes were observed. Babinski negative; spinal fluid normal; leucocyte count, 9,500; urine analysis normal; patient delirious at times.

Dr. G. W. Mackenzie was again called in and he felt that a remaining clot in the left superior petrosal sinus was the cause of the trouble. A second operation was performed on December 9th. The thin intervening wedge shaped piece of bone that remained over the region of the left superior petrosal sinus was removed and the sinus incised. No free bleeding was obtained. The smallest spoon curette was introduced into the sinus and a small clot was removed. A wick of iodoform gauze was placed in the cavity of the sinus. This operation was not followed by any improvement of symptoms.

Ptosis of the left eyelid was first noticed on December 10th. Pupils still equal in size and react normally to light. The patient was delirious at times. The Babinski was questionable on the right side. Small spasmodic twitchings were noticeable on the right side of the face, accompanied by a tendency to grind the teeth. The spasms were tonic in character and seemed to involve the muscles supplied by the seventh cranial nerve. The spinal fluid still remained normal. The mastoid wound looked healthy except for a small area necrosis in the region of the superior petrosal sinus just posterior to the osseous labyrinth and anterior to that which Dr. Mackenzie

removed at the second operation. Whether it was present at the time of the second operation or developed later is a question; however, I am inclined to believe it was present at the time, for the operator hesitated to open the labyrinth for fear of labyrinthine infection following and adding another complication. No changes in either fundus are evident at this time (December 13, 1921.) The patient was in very poor physical condition. Nourishment was taken only with the greatest difficulty.

The morning of the 14th of December the first pupillary change was noted, when the left pupil was found to be slightly larger than the right, but still reactive to light. There was also present a slight degree of exophthalmos of the left eye. Spinal puncture made at this time gave a questionably slight increase in pressure; otherwise it was normal. At 2 p. m. the left pupil was dilated and fixed. The left fundus showed blurring of the disc outline. There were spasmodic seizures of the right side of the face and the right shoulder and arm, but not of the right leg. The patient died at 8:30 p. m.

Postmortem findings: Patient, male; emaciated appearance. The left side of the brain beneath the dura felt fluctuating. The dura was incised and a purulent exudate sponged out. There was no evidence of brain abscess. The brain is normal except for those surface changes found in the presence of a circumscribed purulent meningitis. The sigmoid sinus was observed to have been opened during life. The left superior petrosal sinus was partly opened with a red thrombus extending from about its opening into the left cavernous sinus. The left cavernous sinus also contained a red thrombus.

There is no involvement of the sinuses on the right side.

COMMENTS.

1. Given a case presenting the three cardinal symptoms offered by this patient, namely, (a) trifacial neuralgia, (b) pyemic temperature, (c) the presence of a chronic purulent otitis media, with a discharge having the odor of dead bone, the diagnosis of primary thrombosis of the superior petrosal sinus of otitic origin is justifiable.

2. From the experience gained in this case it is felt that in primary thrombosis of the superior petrosal sinus it is far better to sacrifice the labyrinth in the effort to secure a freer exposure of the superior petrosal sinus than to attempt to conserve the labyrinth at the cost of a less free exposure of the sinus.

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L.

ELECTROCOAGULATION IN THE TREATMENT OF CARCINOMA OF THE LARYNX.

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Diathermy, or electrocoagulation, has long been recognized as an accepted therapeutic measure. Nagleschmidt of Berlin, in 1907, demonstrated the use of high frequency currents in the heating through of tissues. But the special application of diathermy in the field of otolaryngology is a matter of more recent development.

Norman Patterson, in the Lancet in 1919, advocates electrocoagulation of malignant tumors of the larynx in preference to other surgical procedures. W. J. Harrison, C. A. Pfender recommend diathermy in the management of laryngeal carcinoma. On the whole, however, the literature is surprisingly meager in this field, and a complete technic has perhaps nowhere been developed and accurately described.

It is not the purpose of this paper to go into the physics of diathermy nor to dwell at any great length upon the technic of its application to the larynx. A few definitions are perhaps necessary. Diathermy is a means of raising the temperature of tissue by the resistance of the tissue to the high frequency, or D'Arsonval current.

Saberton differentiates between medical and surgical diathermy by the fact that medical diathermy aims to raise the temperature of the tissues within physiologic limits, and surgical diathermy aims to exceed these limits and to produce tissue destruction by coagulation.

There are several special problems to meet in treating the larynx and not the least of these is the question of anesthesia. Local anesthesia is not advisable. Block anesthesia is possible, but cannot be recommended. Of the general anesthetics ether cannot be used because of the great danger of explosion from a possible spark from the electrode. Nitrous oxide and

oxygen are not sufficient to produce complete relaxation, and there is great difficulty in maintaining deep surgical anesthesia. The socalled synergistic anesthesia used extensively by Beck and others in laryngeal surgery is extremely dangerous in these cases, because of the fact that sufficient ether is excreted by the lung to produce an explosion from the accidental sparking at the tip of the electrode. We have been forced to use chloroform at the County Hospital. This anesthetic is surprisingly satisfactory. Its traditional dangers are in a large measure obviated by having a highly skilled anesthetist administer it. There is no danger of explosion, relaxation is complete, it is rapid and the small amount of it necessary to carry on deep surgical anesthesia makes it highly desirable.

Another problem is that of the method of approach. The tumor may be exhibited by doing a laryngofissure. We have condemned this method because of the possibility of dispersion of metastases by cutting into a portion of the tumor. The indirect method of laryngoscopy is not recommended because precision in the application of the electrode is very difficult. There remains suspension laryngoscopy and that is the method par excellence.

The larynx becomes clouded with dense cloudy vapor from the dessication of the tissues during the operation, and it is difficult to see the field of operation. It has been recommended to change the air in the larynx by blowing a stream of air from a pump, but the method found most satisfactory by us is to place a thin metal tube into the larynx, connect this with the suction pump and the steam from the coagulating tissues is thereby removed from the larynx as rapidly as it is formed.

The problem of dosage is still far from solved. It is better in general to err on the side of not doing enough than to do too much, for the operation can be repeated several times, whereas too extensive a coagulation increases the danger of serious hemorrhage when the slough separates. Specifically for a tumor with a diameter, roughly of three centimeters and using an electrode one centimeter in diameter we apply the electrode in two places. With a current of 1200 to 1500 milamperes the electrode is applied in each place for twenty seconds. It is too early to say whether this dosage is sufficient,

but we are inclined to believe that it is not. It will only be possible to determine this by the study of the autopsy and thus far none of our cases has proceeded to that stage. It must be remembered that the average laryngeal carcinoma seen at the County Hospital is far advanced. The early lesion is a rarity. The dosage for a small, early lesion is naturally smaller than that for the lesion which involves the entire larynx. The favorable case is one in which metastastes have not formed and in which the lesion is small.

The immediate results of diathermy are as follows: There is a surprisingly small degree of edema, a preliminary trachetomy is imperative nevertheless. There is a marked and almost dramatic lessening of pain. The bleeding from the ulcer stops. The fate of the coagulated mass is either a sloughing en masse or a fractional sloughing over a period of time. When the coagulation has been radical and extensive there seems to be a tendency to mass sloughing with the attendant danger of severe hemorrhage. When the coagulation has involved only a portion of the tumor, in other words, when the operation has been less radical and when the sloughing has been fractional, the danger of severe hemorrhage is correspondingly lessened, thus making small and repeated dosage more desirable than a complete, radical coagulation.

Electrocoagulation supplemented by radiation is recommended by Pfender. Rhodenburg and Prime have shown that a combination of diathermy (producing a low degree of heat) plus radiation have a greater effect upon malignant tumors than either diathermy or radiation alone. These findings have been confirmed by B. C. Corbus and V. J. O'Connor of Chicago, in the study of the bladder.

Briefly stated, the advantages of electrocoagulation in the treatment of malignant tumor of the larynx are as follows: The operation is bloodless; there is an immediate lessening of pain; there is no danger of dissemination of metastases. The deep penetration of the heat has an inhibitory effect upon the growth of malignant cells (that is, the cells beyond the area of coagulation), as has been shown by Rhodenburg and Prime experimenting with neoplastic cells in vitreo.

Corbus and O'Conor have by animal experimentation found that the body defense against carcinoma metastasis lies in the

formation of a connective tissue capsule The scar tissue that is formed after a diathermy burn is more dense and spreads farther into the surrounding tissue than is the case after any cutting operation. We have here then an extensive reinforcement of nature's attempt to throttle the embryonal cell.

To sum up:

1. Diathermy of carcinoma of larynx is a relatively untried measure. It offers a promising field for investigation.

2. It not only coagulates the tumor mass, but has an inhibitory effect upon the growth of malignant cells adjacent to or at an appreciable distance from the tumor.

3. Chloroform is the logical choice of anesthetic.

4. The laryngeal tumor should be approached by means of suspension laryngoscopy.

5. The amount of current—the length of exposure necessary—requires considerable experience upon the part of the operator to determine, because no set of definite rules of procedure can be formulated in the light of the present limited knowledge of the subject.

6. Radical dosage cannot be recommended.

7. The edema following diathermy is surprisingly slight, nevertheless preliminary trachetomy is imperative.

8. Pain is diminished markedly following coagulation.

9. Diathermy should be supplemented by radiation.

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LI.

VASOMOTOR DISTURBANCES OF THE UPPER AIR PASSAGES AND SINUS DISEASE.

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In the present paper the term vasomotor disturbances of the upper air passages is used to denote the conditions known clinically as vasomotor rhinitis and asthma.

The term sinus disease is here restricted to those alterations of the ethmoid sinuses and of the maxillary antrum which are characterized by edematous hypertrophy arising independently of dental infection. Involvement of the frontal and sphenoidal sinuses may or may not be present. Clinically we find all gradations from polypoid degeneration of the middle turbinate to advanced polyp formation. The maxillary antrum usually contains secretion, in come cases seropurulent in character, with a predominance of mucin. In more advanced stages of the inflammation the periosteum becomes the site of infiltration, particularly in the region of the middle turbinate, giving rise to rarefying osteitis. Bacteriologic examination of the antral fluid is rarely sterile, at times diphtheroid bacilli, and at others the various cocci, such as the pneumococcus, staphylococcus aureus and albus and streptococcus, being present.

It does not appear as yet definitely established whether an individual experiences sensitization to bacteria in the same manner as to proteids entering the system by inhalation or ingestion.

In 1916 the writer offered evidence to show that some anaphylactic individuals give a positive skin test to certain bacterial proteids and that these proteids differ among themselves in their power of causing the skin reaction. Further observation by others, notably by Walker and Rackemann, indicate that anaphylactic individuals may show a very wide variety of bacterial sensitizations.

In a recent paper by Cooke (Journal of Immunology, March, 1922), doubt is expressed whether the group of socalled infectious asthmas can be recognized as a distinct pathologic entity, and the suggestion is made that these may in reality be the result of inhalation of various hitherto unrecognized forms of dust in sensitized individuals.

Without entering into a discussion of this question, the writer wishes to state that in a fairly large number of cases the correlation of clinical with laboratory data seems to lend weight to the belief that sensitization to bacteria exists, and that this sensitization is responsible for the development of vasomotor rhinitis and for asthma in a certain group of cases.

Even if this point be granted, we are not as yet fully clear regarding the nature of such a baterial sensitization. From the standpoint of systematic botany, we might perhaps conceive it to be a sensitization to the proteid of the bacterial body rather than to the products of metabolism of the organism. On this hypothesis the bacterial proteid would be regarded as analogous to the proteid of pollen and of foods. The microorganisms which are recognized as invaders of the upper air passages in acute infections possess the characteristic in common of exerting their influence through the liberation of an endotoxin—that is, a toxic substance existing within the wall of the organism and escaping when the wall becomes sufficiently permeable. This endotoxin is the agent which excites inflammatory symptoms in the invaded individual and gives rise to the phenomena variously of acute rhinitis, pharyngitis, laryngitis and bronchitis. The anaphylactic individual may owe, however, the occurrence of his particular vasomotor symptoms to the specific proteid of the organism and not to the endotoxin. When he is attacked by one of the organisms in question he experiences first the symptoms of a cold in the head or an acute bronchitis from the endotoxin of the organism in question, but these symptoms of inflammation are more or less completely submerged with the liberation of the bacterial proteid.

If this hypothesis should prove correct, it would perhaps afford an explanation of the immediate skin reactions occurring in some cases on the application of bacterial proteids to a scratch, and at the same time indicate that the reactions oc-

curring after the intradermal injection of vaccines may be a toxic manifestation rather than an anaphylactic one. Improvement obscerved in the asthmatic symptoms from the administration of vaccines would then be explained as the result of the establishment of increased immunity to the organisms rather than as a desensitizing process.

Cases have come under my observation where asthmatic symptoms follow a certain type of acute catarrhal infection, and are absent in a subsequent attack of acute rhinitis or bronchitis. As an example, a woman with chronic ethmoidal and maxillary sinusitis, came with a history of nearly constant asthma of several years' duration. Radical operation upon the sinuses was followed by relief of the asthmatic symptoms. Subsequently the asthma reappeared in association with certain acute infections of the upper respiratory tract, but it was found that not all acute infections were followed by asthma. Bacteriologic examination of two attacks with asthma showed a sputum with streptococcus, while on three occasions the sputum from acute bronchitis without asthma showed nearly pure cultures of staphylococcus albus.

There are probably few subjects in which the experience of laryngologists is able to furnish a greater abundance of data than in the one under consideration, but also few in which the evidence of statistics can be more misleading. We are dealing with a number of variable factors, such as the degree of development of the pathologic alterations, the kind and number of microorganisms present, these combining in many different ways to exert an effect upon an individual already himself under the influence of other general forces of a beneficial or harmful character.

Furthermore, the operative measures undertaken in a given case may have been faulty or inadequate, with the result of rendering conclusions inaccurate regarding the etiologic relationship between the sinus disease and the vasomotor disturbance. These remarks apply with more force to asthma than to vasomotor rhinitis, in which less complexity of the various factors exists. Instead, therefore, of attempting to offer a statistical review of all the cases which have come under my observation, it has seemed to me preferable to present for your consideration some of the conclusions to which I have been

led from a study of patients in private practice, upon which I have operated in recent years, recognizing the fact that these are by no means final but are rather of the nature of suggestions to be criticized and probably modified.

Vasomotor disturbances of the regions under consideration may owe their origin variously to the inhalation of foreign proteids, to the ingestion of foods, to the development within the body of infecting microorganisms or to the influence of reflex stimuli. In a given case the causative factor may be one or more of these agents operating singly or in association. Consideration in the present paper is limited to conditions of apparently infectious origin, or to those in which inhalation and ingestion factors, if present, play an intermittent or subordinate role.

It has not been suggested, so far as I am aware, and my observations do not lead me to believe, that anaphylactic disturbances arising from the inhalation or ingestion of foregin proteids, however prolonged, can, in the absence of bacterial infection, give rise to the pathologic alterations of the sinuses above mentioned. We often, it is true, see a hay fever or an occupational inhalation vasomotor rhinitis, as from horse dander or from flour dust, pass gradually into chronic sinusitis, but careful examination of the patient's history will show a series of attacks of acute rhinitis. Since, furthermore, we cannot distinguish, at least from clinical inspection, between the sinusitis unaccompanied by vasomotor symptoms and that occurring in an anaphylactic individual during the periods between the vasomotor manifestations, it seems reasonable to state that the weight of evidence is at present against the production of sinusitis through the agency of inhaled or ingested foreign proteids.

In relation to the question as to whether sinus disease plays an etiologic role in the production of these socalled infectious vasomotor disturbances, we may leave out of consideration to what extent the sinus disease is itself the result of infection. We are concerned simply with the determination whether sinus disease, however established, initiates or influences the vasomotor disturbances, and consequently whether remedial measures directed to relief of the sinus disease will have a corresponding influence on the vasomotor symptoms.

The essential characteristics of vasomotor rhinitis can be determined with relative completeness. We have a pathologic condition corresponding to the familiar wheal produced by the proteid skin test, namely, a serous infiltration and sensory disturbances. Pallor and swelling of the mucous membrane result, together with an outpouring of fluid, and accompanied by sensations of itching with tendency to sneezing.

These anaphylactic phenomena may occur in nasal mucous membranes which are otherwise essentially normal, or they may be superimposed upon tissues already altered by chronic inflammation. In the latter case, a knowledge of the pathologic conditions will enable us to translate the descriptive clinical terms into their histologic equivalents.

Asthma in association with sinus disease may be acute and recurrent or chronic. In the former type we find the individual living for the greater portion of the year in a state of health, but periodically subject to invasion of his mucous membranes by infecting microorganisms. When this invasion develops we find clinically for a few days the symptoms of a cold in the head or of an acute bronchitis, soon, however, succeeded by symptoms of vasomotor disturbance, showing vasomotor rhinitis or asthma. The duration then of the vasomotor affection is like that of an acute, nonanaphylactic disturbance of the tissues in question, and, after recovery, the patient remains free from symptoms until the next invasion.

In the chronic type the individual suffers more or less constantly from asthma, the periods of freedom from symptoms being either brief or absent. Vasomotor rhinitis may coexist with the asthma, although rarely.

In view of the frequent association of sinus disease and of bronchitis, the question suggests itself whether there is an etiologic relationship in the pathologic conditions existing in the regions which predispose them to invasion.

Webb and Gilbert (Journal A. M. A., 1921, p. 714) report a series of cases of chronic bronchitis, in which examination was made of the maxillary and ethmoid sinuses. In a large parcentage of cases sinus disease was found, and operative treatment of the sinuses brought about marked improvement in the condition of bronchial inflammation.

Mullen (Transactions Laryngological, Rhinological and Otological Society, 1919) has shown in animals that lymph absorption from the antrum, whether of bacteria or inert substances, is by way of the submaxillary and internal jugular nodes to the lymph ducts of the great veins, the right heart and the lungs.

In a paper, "Recurrent Influenza in the Nose and Throat," read before this association in 1910, I called attention to a group of cases characterized by acute, recurrent inflammation of the nose and throat, with secondary involvement of the bronchial tubes. The past history is one of annual attacks of inflammation entering the lymphoid tissue of the pharynx and extending promptly to the ethmoid and maxillary sinuses, with involvement of the trachea or bronchial tubes. Purulent discharge appears in the nose and simultaneously cough with purulent expectoration. Examination of the chest shows well defined bronchitis.

If the group of symptoms above described occurs in an anaphylactic individual the symptoms of inflammation may be accompanied by manifestations of asthma which dominate the clinical picture.

It does not appear possible to determine in a given case the relative amount of absorption of bacterial proteids from the mucous membrane of the sinuses and of the bronchi respectively. We may be able to form only an approximate estimate derived from clinical data concerning the degree of development of the sinus disease, the extent of the bronchitis and existence of bronchiectasis. In advanced cases of bronchial disease, especially associated with myocarditis, it would seem of doubtful value to perform a radical operation on the nasal sinuses solely for the purpose of restricting the amount of absorption of bacterial proteids.

The operative management of these conditions of sinus disease in anaphylactic individuals does not differ from that in the ordinary case. It is, however, of especial importance to determine with accuracy the condition of the antra. Evidence obtained from transillumination and X-ray may be misleading, since these methods may show simply the existence of a disparity in the illumination of the two sides. It is possible that

one antrum may be opaque and the other relatively clear, but nevertheless both may have fluid in the interior.

One side may be in a more advanced stage of inflammation with greater thickening of the mucous membrane than the other. In all the cases which I see now, when I have reason to suspect the existence of sinus disease, I do a paracentesis on both sides and have repeatedly found fluid in the antrum which had been pronounced normal by the X-ray.

The antral opening should be enlarged to avoid any possibility of closure and to permit the free escape of the frequently thick viscid fluid. A larger opening is required in these conditions than in the case of simple empyema of the antrum, where the fluid is thinner and more readily capable of flowing out through a small opening.

If the ethmoid regions are affected it is desirable to remove the anterior end of the middle turbinate and curette the anterior ethmoid cells.

Results of operative treatment in chronic vasomotor rhinitis of the type under consideration have been satisfactory in the larger proportion of cases, and the patients have remained free from symptoms except in the event of their experiencing a subsequent attack of acute infection, when a transitory form of vasomotor disturbance may appear. Such a condition is illustrated by the following case:

Mrs. R., 35, was seen November 18, 1920, with a history of vasomotor rhinitis, of one year duration, following a severe cold. Examination showed general pallor of the mucous membranes of the nose, with polypoid degeneration of both middle turbinates. Transillumination was dark on right. All skin tests for foods, pollens and dusts were negative. Both antra were opened through the lower meatus, and the ethmoid cells curetted. The right antrum was filled with thick viscid mucus which showed numerous colonies of staphylococcus albus and ozena bacilli. The vasomotor symptoms disappeared after the operation. One year later the patient had an acute rhinitis, accompanied by vasomotor symptoms, the whole process clearing up, however, within a few days.

Four other cases of essentially similar type have been operated upon in the past two years, with the result of definite relief of the vasomotor symptoms.

In eighteen patients showing sinus disease associated with asthma, of the type under consideration, results of operative treatment have been beneficial, so far as the asthma is concerned, in the majority of the cases. Sometimes after the operation the asthma subsides or remains absent for a while, but is apt to reappear later. Some unsatisfactory results have been manifestly due to imperfect operative technic in the sinus operation, where fluid has reaccumulated in the antra. Washing out the fluid has been followed by again a relief of the asthma. A formation, however, of a large antral opening, which requires no washing, has been followed by a larger number of successful results. In a certain proportion of the cases the change in the clinical picture has been from chronic persistent asthma into an acute recurrent asthma, the patient remaining for a much greater time free from disturbance, but on the development of a cold or acute bronchitis, or after other forms of reflex strain, again experiencing attacks. It has seemed to me probable that the improvement in the chronic asthma is due chiefly to a lessening of the bronchitis and consequent diminution in the amount of bacterial proteid absorbed from the bronchial tract. This view is in harmony with the observations above mentioned regarding the relief of bronchitis by the eradication of sinus disturbance.

My experience with vaccines in these cases of vasomotor disturbance with sinus disease leads to the conclusion that they are of relatively little value so long as the diseased state of the sinus persists. It is only after clearing out the pathologic disturbances in the ethmoidal and maxillary sinuses with the establishment of permanent free drainage in the latter that they are of service.

Where, however, this has been done, the administration of autogenous vaccines, which by the intradermal test excite definite reaction, has appeared to exert a material influence upon the asthmatic symptoms.

In conclusion it must be emphasized that the management of asthma with sinus disease is not alone a problem for the laryngologist but requires the close cooperation of the internist, with constant supervision by the latter of the patient.

Operation on asthmatics with sinus disease should not be undertaken until all possible extrinsic causes, as from dust

and foods, have been eliminated. It happens not infrequently that a case which appears to belong to this category of infectious asthma may in reality owe the disturbance to the presence or a cat or dog in the house or to some article of food in the diet. The observations of Cooke on the influence of house dust are of great importance in this connection.

The degree of development of the bronchitis, the presence of bronchiectatic cavities and association of myocardial affections of the heart must be taken into consideration. If the pulmonary lesions have advanced, little or no benefit may be obtained. In patients with myocarditis an operation may be contraindicated, or at least may be considered likely to afford little or no benefit. As an example, H. B. developed ten years ago asthma and bronchitis following influenza, with annual attacks of bronchitis subsequently. In October, 1921, the asthma became more pronounced, and there was evidence of beginning myocarditis. Both antra were dark on transillumination, and considerable mucopus was present in the nose. Extrinsic causes of asthma were eliminated. Findings were typical of infectious asthma, and an operation would have been performed, except for the condition of the heart, which showed signs of rapidly increasing weakness. The patient died in January from heart failure.

REPORT OF CASES.

1. M. A., 7.—Asthma, four years' standing, associated with frequent colds and attacks of bronchitis. Negative to foods and pollens. Right maxillary and ethmoidal sinusitis. Chronic tonsillitis. Antral fluid showed staphylococcus albus and streptocuccus pyogenes. Radical operation on sinuses. Asthma disappeared and remained absent for nine months, but reappeared on taking cold, lasting moderately for two weeks. Two attacks of asthma occurred during the following year in association with acute infection. No trouble during last six months. Chest now clear.

2. Mrs. E. F. S., 35.—Chronic asthma and bronchitis many years' duration. Marked maxillary and ethmoid sinusitis. Radical operation on sinuses. Staphylococcus aureus in antral fluid. Asthma and bronchitis gradually cleared up. Has been well during past year.

3. W. A., 50.—Asthma and bronchitis 15 years' duration, following severe cold. Ethmoidal and maxillary sinusitis. Operations on ethmoid done repeatedly without relief to asthma. Autogenous vaccine from staphylococcus albus and streptococcus given without result. One year ago both antra opened, left showing considerable mucopus. Bronchitis has improved and for past year has had no asthma.

4. S. W. M., 69.—Asthma many years. Chronic maxillary and ethmoidal sinusitis. Much viscid mucus removed from right antrum showing staphylococcus aureus. Gradual improvement in bronchitis with diminution in wheezing. No symptoms during past year.

5. Mrs. F. W. N.—Asthma many years, recurrent. Maxillary and ethmoidal sinusitis. Radical operation. Improvement in asthma and bronchitis during following three months. Remained then free from asthma until streptococcus bronchitis one year ago, lasting one month. Since then free from disturbance.

6. M. A., 40.—Asthma several years with bronchitis. Many operations have been done for removal of polypi. Marked inflammation of ethmoid bones, opacity over antra. Radical operation on sinuses. Slow improvement during following six months, attacks of asthma, however, being less frequent and severe. Seen two years after operation, having had no asthma during previous year.

7. Mrs. G. E. K.—Thirteen years ago chronic right maxillary and ethmoid sinusitis observed. Radical operation done. Now right frontal sinus shows considerable secretion. Intranasal operation on right frontal sinus. Four months later asthma began gradually, increasing, in association with involvement of left antrum. Antral fluid showed staphylococcus aureus and albus. Autogenous vaccines from these followed by no relief. During following year asthma became more trodblesome, and infection of right antrum was observed. In May, 1920, both antra were opened freely, and polypoid enlargement of left middle turbinate removed. Has had no asthma for two years since operation.

8. Miss E. F. M., 70.—Seen first fourteen years ago. Asthma many years' duration. Repeated operations on ethmoid sinuses without relief. Sinuses were then curetted with-

out improvement of asthma. During next eight years no essential change in asthma; chronic bronchitis was noted in 1913, the right antrum became infected, and was tapped with removal of much pus. Autogenous vaccines were administered without relief. Five years ago tonsils were removed and showed large amount of detritus. Chest examination showed extensive chronic bronchitis with a moderate degree of myocarditis. Asthma still intermittently troublesome. No definite result could be shown following the various operations.

9. D. D., 30.—Bronchitis two years following severe strain during war. Marked ethmoid sinusitis. No involvement of antra. Curettage ethmoids was done nine months ago. Antra opened. No fluid found. No relief following operation.

10. Mrs. C., 50.—Seen 17 years ago. Asthma many years' standing with chronic bronchitis. Marked ethmoid sinusitis. In 1906 large amount degenerated ethmoidal tissue curetted on both sides. Asthma has persisted more or less constantly since. It has not been possible to obtain the consent for operation on antra, although these are evidently infected, as shown by X-ray. Now has extensive bronchitis with myocarditis.

11. Mrs. C. H. M.—Asthma ten years, more or less constant. Some operation on sinuses was done ten years ago, with removal anterior ends middle turbinates. Transillumination at time of examination dark both sides. Curettage of ethmoid cells with large opening into each antrum. Antral fluid showed streptococcus. Asthma has been troublesome since operation during past six months following operation, but as patient lives at a distance it has not been possible to follow condition accurately.

12. Mrs. E.—Seen August, 1914, history of infection right antrum last spring, with asthma more or less constant, conditions persisting throughout summer and winter with each cold. In August, 1915, both antra were tapped, large amount of fluid removed, asthma nevertheless persisting. In October antra were again opened, and large amount viscid mucus removed from left. Middle turbinotomy. Improvement followed lasting several months, asthma again returning. Sinuses repeatedly washed with temporary relief. In 1916 no asthma throughout summer. Both antra free. In winter of 1916

asthma reappeared, with evidence of myocarditis. In January, 1916, large free openings made into each antrum, which showed a quantity of viscid mucus. Cultures of this fluid showed diphtheroid bacilli, few streptococci and occasional colonies of staphylococcus. Asthmatic attacks persisted throughout winter of 1918 and 1919, becoming extremely severe in March, with progressive heart failure, from which the patient died March 16th.

13. Mrs. G. Y. B., 40.—Asthma and chronic bronchitis many years. Chronic tonsillitis Chronic ethmoidal and maxillary sinusitis. Tonsillectomy and radical operation on sinuses. Staphylococcus albus in antral fluid. Asthma disappeared, remaining absent for three months. In past six years, five short attacks of mild acute bronchitis and asthma.

14. Mrs. A. P., 35.—Asthma several years, generally persistent. Chronic ethmoidal and maxillary sinusitis. Streptococcus in antral fluid. Radical operation. Asthma became progressively less severe during following two years. For past three years no asthma.

15. Mrs. J. B. P., 35—Asthma several years, generally persistent. Chronic bronchitis Chronic ethmoidal and maxillary sinusitis. Antra opened. Cultures of antral fluid showed staphylococcus albus and streptococcus pyogenes. Since operation five years ago attacks of asthma infrequent, usually brief and following colds or overwork.

16. Mrs. M. F. R.—Frequent attacks asthma and bronchitis. Chronic ethmoidal and maxillary sinusitis. Radical operation. Since operation three years ago no asthma.

REMARKS.

In all the above cases skin tests for foods and pollens were negative. A few showed positive reactions to dusts such as horse dander and cat hair, but the influence of these factors upon the asthma was definitely eliminated.

The statement, "no asthma," denotes that the patients have been free from symptoms of the type which had previously troubled them. Excessive fatigue, nervous strain or exposure to certain dusts, in patients sensitized to such proteids, have been sufficient to excite a temporary recurrence of the asthmatic symptoms.

CONCLUSIONS.

A group of vasomotor disturbances characterized by vasomotor rhinitis or by asthma exists standing in relation to chronic infection of the nasal sinuses. In vasomotor rhinitis the sinusitis is probably the direct cause of the disturbance. In asthma of this type, it is not wholly clear what part in the production of the disturbance is played by the presence of bacterial infection in the sinuses. There is reason to believe that the sinusitis has an etiological relationship with the associated bronchitis. Relief of the sinusitis is usually followed by diminution in the bronchitis and asthma, unless the bronchial lesions are old and extensive or associated with myocarditis. Operative treatment of the sinusitis consists in adequate removal of diseased conditions in the ethmoid bone with the establishment of large openings into the antra to permit the spontaneous evacuation of the antral fluid.

LII.

BARANY'S PRIORITY.

By Irving Wilson Voorhees, M. D.,

New York City.

In the Acta Laryngologica, Vol. III, Fasc. 4, which has just come to hand (June 1, 1922), the first article is entitled, "Barany und die Wiener Universität." Five gentlemen, namely, Burger of Amsterdam, Forselles of Helsingfors, Holmgren of Stockholm, Schmiegelow of Kopenhagen and V. Uchermann of Kristiania have taken up the cudgels for Dr. Barany and have set down all the available facts regarding the controversy which has waged about Barany's name ever since the recognition of the prize award by the Nobel Prize Committee. Rumors have come to us on this side that there was a good deal of dissension in Vienna at the time of Dr. Barany's election to the Upsala, Sweden, faculty, but most of it has been hearsay and in the nature of "backstairs gossip," therefore, it seems only fitting to place before the many old students and admirers of our distinguished confrére what this tempest in a teapot is all about.

A short time after Docent Barany had received the Nobel prize for his essay on the Physiologie and Pathologie des Bogengangapparates beim Menschen. (1907), Professor Victor Urbantschitsch recommended Dr. Barany to succeed to the chair of Otology in Vienna University. To the astonishment of those who knew about it, this recommendation was not ratified by the Vienna medical faculty, but no explanation was forthcoming until contumely and canard reached out so far that Dr. Barany was obliged to take recognition of the state of things and ask for an explanation. It then came out that the attack was in secret except for the open attitude of Herr Tandler, Professor of Anatomy. Barany was unable, however, to get any "official" information from the Dean of the University, nor was he allowed to come before the so-called "Senate" to answer such charges as had become the

gossip of two continents. He then took the matter higher up, to the Minister of Education, who seems to have succeeded in getting the matter up for discussion. The specific charges accuse Barany of bad faith in claiming originality, and willful carelessness in the use of scientific material. Specifically he had said that certain disturbances in the musculature of the torso are localized not in the hemispheres of the cerebellum, but in the worm. A patient on whom he had made this diagnosis died and he presented the autopsy findings which showed an abscess in the worm as he had said. He failed, however, to mention the fact that there was also an abscess in one of the hemispheres, although the cut brain section showed it, and it was so stated in the published report of the case. The history of this patient with all of the findings had been presented at the congress in London, and later to his confréres in Vienna.

Another charge had to do with "Gross negligence in making false statements as well as attempting to increase his own prestige at the expense of the work of other investigators." In Barany's defense his coadjutors cite two discoveries that are most certainly his: (1) Caloric nystagmus; (2) influence of position of the head upon the falling reaction or direction. The accusers deny the strict interpretation of these statements and say that he took the work of Breuer and Ewald and published it as his own. His defenders say that such work was basic, to be sure, but Barany carried it much further, established it and made it practical, especially as to the facts relating to eye and head nystagmus, about which Breuer said nothing. His defenders say that otology is indebted everlastingly to Barany for the following:

1. That we can produce a caloric reaction in a sound, healthy human ear with warm or cold water.

2. That this reaction is constant and can be definitely described and measured.

3. That he first described "contrary nystagmus" produced by heat or cold.

4. That he first described the changes or reversal in the direction of movement by altering the position of the head 180 degrees.

5. That he first called attention to the dependence of nystagmus upon position of the head in contradistinction to rotational, galvanic or pressure (mechanically produced) nystagmus.

6. That he demonstrated caloric nystagmus to be the result of endolymph movement—a concept which was mentioned by Breuer among other things, but first set down by Barany as the true explanation.

Furthermore, as to the charge that he did not give credit to other investigators there is Politzer's Geschichte der Ohrenheilkunde, in which Barany mentioned in his article the names of Hitzig, Baginsky, Bornhardt and Breuer, merely stating that he, himself, had discovered and applied the laws of caloric nystagmus, not the original facts. In 1906 he published a comprehensive work on the results of his "investigation into the reflex rhythmic nystagmus arising from the vestibular apparatus of the ear and its associated symptoms," in which he acknowledged that the facts came from Purkinje's work, also those of Breuer, published in 1874; but, unfortunately, in his 1909 report before the Budapest Congress he forgot to give credit a second time to Breuer which made it appear that he, himself, had made the discoveries, thus playing directly into the hands of his enemies. This was, indeed, a grievous error which he has tried to acknowledge and to apologize for, but which will not down.

Out of the maze of vituperation and invective the character of Breuer looms up large, and he absolves Barany from all and every alleged attempt to deceive. In a letter dated April 30th, 1921, he says: "I have found two reprints of my first labyrinth work, published in 1874, and send herewith one to you and one to Hofrat Meyer. You will see that I had not then observed and had not described the falling reaction resulting from change of head position. I cannot accept priority of this nor of the caloric reaction in order to injure Barany. I would not do so even if I had cause to complain of any grievance. So much the better is it that my refusal to claim priority rests upon the supreme truth. Therefore, if you are interested, convince yourself of the veracity of my statement—if not, throw the reprint into the fire or return it to me."

There are other statements from the five learned gentlemen which go to prove that the accusations do a great injustice to Barany, but enough has been cited, perhaps, for the American reader to draw his own conclusions. In closing the five professors say: "We honor Barany as the first man since the days of the great pioneers in otology who has opened up a new epoch in clinical experience, and we protest against the attempts of his hidden enemies who would belittle the significance of his scientific contributions and would besmirch his honor and integrity."

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of February 6, 1922.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, IN THE CHAIR.

PRESENTATION OF CASES.

DR. H. C. BALLENGER presented a patient with two

Canine Teeth Which Had Extended Up Into the Septum.

She had come in complaining of nasal discharge and crusting and these teeth were found incidentally. She gave a history of all the first teeth having to be pulled with difficulty. Although the girl was twelve years old, all of the upper molars were still the first teeth. The Wassermann reaction was positive. Apparently of the hereditary type.

DR. JOHN A. CAVANAUGH presented an

Instrument for Removing the Anterior Wall of the Sphenoid.

This instrument was an electrically driven burr, which is hooded so that there is only one cutting surface. There is a mark on the handle so that one can tell at all times just where the cutting surface is located. With this instrument the thick lower wall can be removed, and better drainage obtained than can be had by any biting forceps.

DR. G. W. BOOTH presented a

Forceps for Removing Peanuts and Similar Fragile Bodies From the Bronchus.

The instrument was very light and preserved the sense of touch.

DR. NORVAL H. PIERCE addressed the Society on

"A Skiagraphic Study of the Temporal Bone in Relation to Normal and Abnormal Pneumatization."

Dr. Pierce stated that since 1917, when Wittmaack's book appeared in Germany, he knew of no work that has proved or disproved his results. Nearly five years have elapsed and

nothing has yet been published that would disprove his conclusions.

Wittmaack divided the pneumatization of the temporal bone into three stages; the first stage covers the first year or thereabouts and consists in the pneumatization of the cavum and the antrum. Up to this time there is no pneumatization of the mastoid process. At the end of the first year and the beginning of the second pneumatization begins in the mastoid, and at the end of the fifth year should be complete under normal conditions. These cells, the concentric and excentric, form the antrum. The third stage lasts through life. As long as a man lives, if there is no arrest in pneumatization due to inflammation which occurs in the first years of life, his mastoid progressively pneumaticizes and this process occurs as it does in the original process, by the invasion of narrow spaces from preexisting pneumatic spaces. This process has been discovered in men as old as seventy.

The pneumatization of bone is really of secondary importance in the process to the changes which take place in the mucous membrane. In order that a pneumatic space may be formed, it is absolutely necessary that the epithelium shall gain entrance to that space. Otherwise, no space can be formed. In many of these spaces there is the narrow space invaded by this submucous myxomatous tissue, but unless the epithelium penetrates into it this myxomatous tissue changes, after it causes the absorption of the marrow proper, into connective tissue, and when this occurs, instead of the marrow spaces being absorbed, there is apposition of this connective tissue and we have the process of sclerosis occurring. In this way the sclerotic bone originates. It is not due to long-continued suppuration but to a failure in normal pneumatization of the mastoid process. Although the red marrow spaces stand at hand ready for the invasion of the myxomatous tissue, nothing of the kind takes place.

What has formerly been called tubotympanal catarrh depends on the arrested pneumatization, and we gain knowledge by Roentgen study of the condition of the mucosa, because when there is arrested pneumatization we know of necessity that there must be a certain type of submucous tissue,

because the condition of the submucous tissue is what has arrested the pneumatization. When certain changes have taken place the epithelium is unable to go down into the bone in order to pneumatize it. In all these cases we have a hyperplastic condition.

In the acute inflammation these cases never come to a frank mastoiditis. That is easily understood, because the anatomic parts are not there for the production of this picture; namely, large, well distributed pneumatic spaces.

In the rather arrested pneumatization there is an entirely different picture. We do not find the spaces that are found in the perfectly pneumaticized mastoid. Instead there is a large mass of bone, and if cells are formed those cells have very thick walls, which are very much more resistant to the softening process, naturally, than the very thin, delicate walls of the pneumatic spaces in normally pneumaticized mastoid bone. Not only that, but this hyperplastic tissue is a protection in itself from the bone. In the normal case there is a very thin mucosa, and in the arrested pneumatization a very thick hyperplastic mucosa. In these cases we have the chronic, running ears after this invasion, not a frank mastoiditis. The discharge comes from the tube, from the cavum and from the antrum, but the bone itself seldom softens.

Dr. Pierce disagrees with Wittmaack in that he has found numerous cases of partially arrested pneumatization in which symptoms have developed which threatened the life of the patient because of the invasion of neighboring parts, principally the sinus. These are only in partially arrested pneumatization. In the totally arrested cases it is rare that an involvement is found of either the labyrinth, the cranial cavity or the sigmoid sinus by extension of the softening process, as would occur in very slightly arrested pneumatization or in normal pneumatization. In those cases we do have involvement of the sinuses, but this is by way of the vascular system. The abnormal pneumatization of these vascular remnants between the cells and the blood vessels or meninges occurs and one must give a very careful prognosis in these cases. The vascular connection is probably beyond operative skill.

Dr. Pierce believed these very valuable conclusions could be drawn from skiagraphs. We can tell whether the mucosa is thin. We can tell whether pneumatization is complete. We can tell whether the pneumatization is complete with hyperplastic mucous tissue. We can tell whether there is incomplete pneumatization with a hypertrophic mucous membrane. We can tell whether there is incomplete pneumatization with fibroid mucosa, and all of these have great bearing as to our prognosis and our treatment. For instance, if we have a running ear, without cholesteatoma and with greatly arrested pneumatization, we may know that such a case does not of necessity require operation. That discharge does not come from bone because we know that the bone in these cases is highly resistant. We know that the discharge comes from the highly plastic mucous membrane. We know that in these cases softening is not likely to occur, and if we do operate, the only thing to consider is a radical operation. If we do not do a radical, then we do not drain the cavum, and if we do a radical, then we cannot remove the thickened, hyperplastic tissue of the cavum and the tube. And unless we are quite sure of our technic of closing the mucosa in the cavum we still have an ear that discharges as much as before, and we will have operated on a case that was not dangerous, and will not have improved the patient at all.

DISCUSSION ON PAPER OF DR. NORVAL H. PIERCE.

Dr. Joseph C. Beck said it was a great pleasure to again hear this subject presented by Dr. Pierce. At the time of his previous presentation he was perhaps the only one who was in possession of Wittmaack's book, and in the discussion Dr. Beck mentioned the importance of radiographic study in connection with the work. He now agreed with Dr. Pierce that it should be of the greatest value in connection with the study. He did not believe that Dr. Pierce thought that by means of the X-ray one could determine exactly the type of mucous membrane, but only by inference from the bony changes which were present. In a bilateral, suppurative process it would be difficult to utilize this subject, for when both sides are involved it is difficult to see just what kind of a process is going on.

After receiving the book of Wittmaack's and discussing it with several authorities in this country, Dr. Beck had written and asked Dr. Wittmaack what he would think of having his book translated into English. Dr. Wittmaack was anxious to have this done and had given permission for the plates to be used in this translation, and the work is now being done by Dr. Bigelow of Providence.

Dr. Beck was sure that all saw the importance of the work as brought out by Dr. Pierce.

Dr. J. Holinger was of the opinion that this explained a number of points that are important in the formation of cholesteatoma. In cholesteatoma there are usually small cells and a small antrum, not larger than a bean. Dr. Holinger did not like the expression, "sclerosed mastoid," but preferred solid or "eburnized." He believed the sclerosed mastoid might be confused with otosclerosis, with which it had nothing to do. He believed that in the cases that used to be called dangerous mastoids, with a thick cortex and a small antrum filled with cholesteatomatous masses and debris, the antrum often became enlarged through absorption, not of inflammatory nature, but purely pressure absorption on account of enlargement of the cholesteatomatous masses. After the operation the horizontal canal was found opened: One would find a tiny groove on the inner wall of the aditus which was nothing but the opened horizontal canal. The process in these cases is always very slow. These points were offered here in explanation of a case shown by Dr. Holinger some time ago, where a man in perfect health suddenly showed protrusion of the posterior superior wall of the canal and a small cholesteatomatous mass was expelled. This opening closed, but after a few weeks another of these masses appeared, and this was repeated five or six times. He now had another patient in whom the same process has been going on for a year. Suction helped to deliver the masses. There was no sign of fresh or old inflammation and no discharge. After that the wound closed and the bulging disappeared. Dr. Holinger thought the explanation given by Wittmaack and Dr. Pierce was much more plausible than the other explanation—that perhaps as a child this patient had a suppuration in the antrum and immi-

gration of the epidermis from the external canal into the antrum, causing displacement of the lining of the antrum and afterwards closing the tympapic membrane over it.

As to the original cause that Wittmaack gives for the whole process of arrested pneumatization—the entrance of amniotic fluid through the eustachian tube into the middle ear during birth with consequent arrested pneumatization and arrested change of the cuboid and cubic epithelium into the normal flat epithelium, Dr. Holinger thought this a little bit far-fetched. In every newborn baby the lungs, the nasopharynx and windpipe are filled with amniotic fluid. The statement that the inflammation caused by that amniotic fluid produces this whole process, Dr. Holinger thought needed confirmation.

Dr. G. W. Boot thought that instead of the chronic, catarrhal ear being the result of the myxomatous tissue it might be the other way around and that because of the air not entering the cavity the submucous tissue was not absorbed.

Dr. Alfred Lewy thought Dr. Pierce was taking a very pessimistic view in saying that a child who gets amniotic fluid into the ear with resulting defective pneumatization of the mastoid was due for a life of ear difficulties. He also removed from the small list of conditions leading to deafness that we have heretofore thought curable, tubotympanic catarrh, so that nothing was left that we can treat successfully but acute inflammatory conditions. Of course, his observations must all be subjected to clinical checking up, like the cases mentioned by Dr. Beck.

Dr. Lewy had recently had under observation what appeared to be a thoroughly pneumatized mastoid, which, according to the X-ray picture, had cells extending into the zygoma and even into the squama, but on operation no cells were found in the zygomatic or squamosa regions, merely diploetic bone, so we must be careful in interpreting our X-ray pictures.

He believed that it would take a great deal of clinical investigation to determine whether the failure of pneumatization was always to result in incurable ear conditions, or was always the underlying cause.

DR. G. E. SHAMBAUGH called attention to the symmetry usually existing between the mastoid process on the two sides. Where one is small and undeveloped, the other as a rule is small and undeveloped. Where one is large, fully pneumatic, a similar condition exists on the opposite side. Where one shows only a partial pneumatization, exactly the same type of condition is to be expected on the opposite side. For these reasons he was not ready to accept the view that the extent of pneumatization or even its complete absence is necessarily always dependent upon the otitis media. On the other hand, he was willing to accept the view that chronic suppurative otitis media occurring before pneumatization has taken place prevents this from developing. The reason why nonpneumatic sclerosed processes are found in connection with chronic discharging ears is due often to the fact that pneumatization is prevented because of the chronic suppuration. On the other hand, he believed that in acute otitis media with an involvement of the pneumatic spaces, the membrane lining the air cells undergoes inflammatory thickening, leading eventually to the genuine sclerotic process. He has sections of such a mastoid process showing these inflammatory changes.

Dr. Shambaugh also called attention to excessive pneumatization as the direct opposite of the condition discussed above. Pneumatic cells develop at a distance from the confines of the mastoid process. For example, around the tympanic orifice of the eustachian tube internal to the digastric groove and toward the apex of the petrus bone. He has seen cases where the skiograph showed pneumatization spreading back to the torcula.

DR. JOHN A. CAVANAUGH said he had been doing work along the line of otosclerosis and as Dr. Pierce was showing the pictures, Dr. Cavanaugh recalled some points in connection with his own work.

Dr. Cavanaugh thought the marrow spaces which Dr. Pierce pointed out resembled the histologic specimens of otosclerosis. The anterior part, where we recognize the beginning of otosclerosis, he believed, showed an arrested stage. The anterior part of the oval window, according to Huxley, is developed from the opisthotic osseous centers, and this Dr. Cavanaugh

thought is arrested in the process of development, and later on, owing to something, whatever it might be, starts the production of bone cells. He has not had any human specimens to work with because they are hard to get, but has been working on young cats, about one month old. Dr. Cavanaugh thought some of the pictures shown were very similar, and that it might have some bearing on the cause of otosclerosis.

Dr. Robert Sonnenschein said that Dr. Pierce's reference to the absence of a frank mastoiditis in the cases of arrested pneumatization was well borne out in a case under observation for several years, a patient Dr. Pierce had also seen several times. This patient was a boy of seven; at four years he developed an otitis media, had another attack at five and another last year. Each attack begins with very mild rhinopharyngitis. A parencentesis is done and a diffust discharge begins. This condition persists for about ten weeks. Ordinarily one would expect a breaking down of the trabeculae but this does not occur. After each onset the patient entirely recovers until the next attack. The pictures taken during the last one showed the arrested pneumatization.

Dr. Sonnenschein thought it was unfortunate that Dr. Pierce could not show all of his X-ray plates and thanked him on behalf of the Society for his excellent presentation and for so extensively studying the subject.

Dr. Pierce (closing the discussion) said that the picture he showed of a man of sixty might well be considered that of a child of a year and a half. There was not a single area in the bone which one could possibly imagine to be a pneumatic space that had been closed up by sclerosis.

He agreed with Dr. Holinger that it was well to keep away from cholesteatoma because this was an entirely different problem. The explanation of Wittmaack was the most intelligent explanation we have at present.

As to the causation of this trouble, the entrance of amniotic fluid into the tube acting as a foreign body, Dr. Pierce thought was the best explanation we have at present, and it is known that several observers who have performed thousands of autopsies on infants have found this condition present. Long

before they associated it with arrested pneumatization they found all these histologic changes present, and Wittmaack had said that on account of the small size of the children's clinic at Jena it was difficult for him to find a normal pneumaticized bone—that they were practically all types of arrested pneumatization. All observers have put the process of otitis media neonatorum at from 85 to 90 per cent of infants. Zuckerkandl's description of the three types of mastoid which have hitherto been regarded as normal, the mixed type, the sclerotic type and the rheumatic type, gives statistics which are not far off from the statistics of Wittmaack. The infant mortality must also be considered. Many of them do not grow to manhood. Insurance statistics and vital statistics might clarify the question somewhat as to how many children die in infancy. Dr. Pierce said he was not an advocate; he did not believe that the causation had been proved. As Dr. Lewy said at the last meeting, it might be due to abronmal secretion of the endocrins. The ears are sterile in the great majority of cases and the bacterial infection is due in the great majority of cases, where the inflammatory element is introduced which produced certain changes, to the pneumococcus.

Dr. Shambaugh had called attention to the symemtry of the mastoids, but Dr. Pierce could prove by at least four of his plates that a symmetry occurs with relative frequency. In one case there was partially arrested pneumatization and in the other ear almost totally arrested pneumatization.

As to the entrance of air into the cavum causing this disturbance, this was unthinkable to him. This process takes place in birds as well as in humans and he believed this did not enter into the consideration.

As to Dr. Cavanaugh's trying to mix this proposition up with otospongiosis—not otosclerosis, which Dr. Pierce thought should be stricken out of medical literature—he believed otospongiosis had nothing to do with sclerosis. That is changing the bone into spongy tissue, and Wittmaack has been particularly careful to say that this process has nothing whatever to do with otospongiosis.

Dr. Frank J. Novak, Jr., presented his inaugural thesis, entitled:

"Electrocoagulation in the Treatment of Carcinoma of the Larynx."*

*See page 878.

DISCUSSION.

Dr. A. B. Kauffman emphasized the advantage of diathermy in the treatment of carcinoma in general. In considering the reasons why carcinoma recurs, apart from the factor of stimulation, cutting and removal of sections which often causes rapid growth of the malignant structure, the question of incomplete surgical removal must be considered. One important factor about diathermy is that it acts on the socalled migratory cells and thus reduces the possibility of recurrence. One of the reasons for recurrence of the malignancy is the infiltration of the carcinomatous cells into the adjacent normal tissue, where they are found in the lymph channels and other locations. Dr. Kauffman believed this method was of particular advantage in this respect. Malignant cells are known to be destroyed by heat at about 50° C. and the normal by 60° C. Using this as a working basis one has, in addition to the zone of coagulation, another zone at the periphery where the heat is raised to about 50°, which will inhibit the growth of the cells in the normal tissue. Dr. Kauffman thought the work of Dr. Corbus was confirmed by that of Clark, who suggested preradiation to inhibit the action of the migratory carcinomatous cells, as complementary to diathermy. The depth of heat penetration is found to extend 6 to 8 centimeters beyond the depth of the zone of coagulation, and accounts to a certain extent for the success that can be hoped for in the treatment of carcinomas in general.

Dr. Kauffman thought one of the dangers lay in using too strong a current and there was also the danger of attacking the larger vessels and nerves in the vicinity. In malignancy of the larynx great care must be exercised not to use too great a current on account of the carotid, because it is known that the heat penetration is far beyond the actual contact. One must be very familiar with the technic used and with the amperage which may be used with safety on the different tissues. Fat is very penetrable and the carti-

lage is more resistant, thus preventing the penetration of heat into the tissues which appear healthy and normal, but which in the lymph tissue and ducts contain the migratory cells which later may become veritable germinal centers for recurrence of the malignant cells.

Dr. Joseph C. Beck thought that anyone who wished to do something for carcinoma in any part of the body could feel encouraged from this presentation. When Nagleschmidt first presented his book Dr. Beck was much interested in the method applied and was glad Dr. Novak had not mentioned his publications in 1907, because he knew now it was not surgical diathermy, but simply charring the parts, as he then understood it. Dr. Beck believed Dr. Novak's work would be of great value and they expected to apply it. He had asked Dr. Orndoff to be present and listen to the paper so that the method could be used in their work. He agreed with Dr. Novak that time will show just what can be accomplished.

Dr. Beck again presented the man who was shown at the January meeting to demonstrate the improvement in the voice with the use of the artificial larynx of Burns. He believed that if a patient lived through a laryngectomy and could use this appliance so that he could talk intelligibly he should be satisfied with the result.

Dr. Alfred Lewy said he had a limited experience with diathermy in carcinoma of the throat, although none of his four cases were in the larynx. He had been afraid to undertake this work in the larynx because of the supposed danger of aspiration of infection from the slough which follows this operation. His method was a little different from that of Dr. Novak. He had used hyoscin, morphin with ether anesthesia, using very little ether, which was always withdrawn for a minute or two before the operating electrode was inserted, and he used a large needle instead of a flat electrode. This had the advantage of requiring less amperage in order to obtain the same results. An area of about one centimeter in diameter around the needle is coagulated in perhaps two or three seconds instead of twenty, so the question of vapor obscuring the sight hardly arises.

The relief from pain in one case was marvelous. The patient had hardly been able to swallow even liquids for about a month, but the day following the operation he was able to eat a square meal. The three other cases were practically not relieved until after the slough separated.

Dr. Lewy believed the fear of hemorrhage was not altogether justified. His cases had been in the region of the tonsils, and side of the tongue, and he had to work rather close to the large vessels of the neck. He had been perhaps rather bold, but he believed that under the circumstances in dealing with malignancy which offered no hope unless completely destroyed we were justified in taking this risk, as death by hemorrhage was perhaps to be preferred to death by cancer. However, there was no such trouble in any of his cases.

In January this year he had shown a patient who was operated some sixteen months before and was in good health with no sign of recurrence or metastasis. Another patient had no recurrence in situ, but died of metastasis after nine months of complete relief so far as the throat was concerned. Of the other two one had been extensively rayed before trying the coagulation but without effect. The other, who had a growth involving the side of the tongue and floor of the mouth, went on from bad to worse.

Dr. Lewy knew of four machines on the market; probably there were more, but he did not know the advantages of each, except that the newer ones were capable of much higher amperage and better control. He believed this method would prove a valuable addition to our armamentarium in the treatment of carcinoma, especially of the mucous membranes. He asked Dr. Novak how far back his cases dated, and whether or not aspiration pneumonia had occurred in any of them.

Dr. Samuel Salinger believed that Dr. Novak deserved much commendation for taking an interest in these cases, most of which were absolutely beyond the hope of surgical intervention. He had seen several of them at the County Hospital and thought Dr. Novak was very modest in withholding his results obtained so far. He knew of two cases

in which the progress of the growth was arrested, the patients were much more comfortable and had begun to take on weight. Dr. Salinger thought this particular method of attacking malignant growths in the larynx was more applicable to the advanced cases than the early ones. He still held to surgical intervention in the cases that are intrinsic. He believed there was a great danger of hemorrhage from diathermy in the pharynx and knew of one case that came to a fatal termination shortly after treatment. Whether the diathermy was directly the cause, or whether the malignancy had already reached the large vessels, he could not say.

Dr. Norval H. Pierce asked what the probabilities were of stricture of the larynx developing. If it was true that diathermy caused the production of connective tissue, was there not danger of stenosis occurring even without stopping the growth of the carcinoma?

Dr. Frank Novak, Jr., (closing the discussion) said that the cautery was an accepted method but its limitation lies in the fact that its heat penetration is very slight. The high frequency current has not these limitations but has great penetration.

As to Dr. Lewy's fear of attacking the larynx because of the danger of aspiration pneumonia, so far no case had developed an aspiration pneumonia. He had three cases of carcinoma of the tonsil, four of the tongue and four of the larynx, and one of the palate. Regarding the use of the needle instead of the electrode he used, there is a greater possibility of carbonization with the needle, with a resulting interference with the penetration of heat. His cases had been under observation for about six months.

Dr. Novak did not agree with Dr. Salinger that the method was more applicable to the late than the early cases, because the late cases of carcinoma are hopeless from any standpoint. Metastases always have occurred and treating the original site of the carcinoma only prolongs the patient's life. In his opinion the most suitable case is the early one, where the growth is circumscribed and limited and any invasion of the lymphatic tissue in the vicinity of the tu-

mor by the migratory cells can be coped with by the deep penetration of heat.

Replying to Dr. Pierce, he thought his remarks were interesting, but so far he had not seen any stricture. The scar that is formed is smooth and if one can go by the results obtained by the urologist in work in the bladder, the possibility of stricture is remote. They have found that they can use this method in bladder in the immediate vicinity of the ureters without causing any stricture. The opening persists and no stricture results. He thought it would be possible to tell more in this regard at a later date.